The New England
Journal of Medicine

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Volume 236 MARCH 20, 1947 Number 12

SECOND-DAY ENCEPHALOGRAPHY, WITH PARTICULAR REFERENCE TO THE SIZE OF THE VENTRICLES*

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THE literature contains a comparatively small number of publications concerning the appearance of the ventricles on the days following encephalography, although it is quite probable that a number of investigators have been interested in such follow-up studies without publishing them. Of the published reports, some were confined to the rate of absorption of air after encephalography,[1, 2] whereas others emphasized the importance of second-day studies for the demonstration of porencephalic cysts.[3, 4] Only a few observations were concerned with the size of the ventricles during follow-up studies, and those were quite contradictory. Schott and Eitel,[5] as well as Reinberg,[6] mention dilatation of the ventricles in later films as if it were a routine finding. This broad statement is made without any supporting evidence; no actual encephalograms are reproduced to illustrate this so-called "routine second-day enlargement." The explanation of Davidoff and Dyke[1] — that these authors mistook the better visualized anterior horns for enlarged ventricular bodies — appears quite plausible. Friedman, Snow and Kasanin[7] state that the shape of the anterior horns usually changes on the second day from the butterfly pattern to a more or less rounded form because of transitory internal hydrocephalus. This observation can apparently be explained on a basis similar to that mentioned by Davidoff and Dyke,[1] who state that there is no enlargement of the ventricles on the second day. Evans[8] reported follow-up studies in 39 children, the majority of whom showed a slight increase in the size of the ventricles on later examination. In 7 cases there was a more striking increase in the size of the ventricles on the second day. Paul and Erickson,[3] in a recent publication concerning second-day studies in 78 patients, found the ventricles usually decreased. Seven of the patients, however, showed an increase in the size of the ventricles on the second day. It was interesting to find that 4 of the 7 cases were post-traumatic, whereas 2 were cases of idiopathic epilepsy and 1 was a case of Alzheimer's disease.

During our encephalographic studies in a series of head injuries, some abnormalities were found that led to a systematic examination of the skull on the days following the lumbar insufflation of air. We found definite enlargement of the ventricular bodies in a third of the group, the enlargement being extensive in some cases.

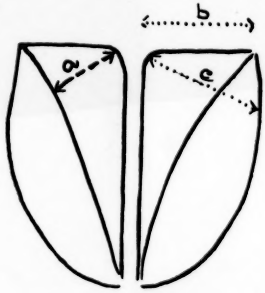

FIGURE 1. *Ventricular Measurements in the Anteroposterior View.*

The measurement of the width of the ventricles (a) *was considered the most important of the measurements; it is the shortest distance between the lateral wall of the ventricular body and the superior medial corner of the ventricle.*

MATERIAL AND METHODS

Patients Examined

Among a large group in which encephalography was done, second-day studies were performed in 67 cases. In 60 cases, in which examination on the first

*Presented at the annual meeting of the Massachusetts Medical Society, Section of Radiology, Boston, May 23, 1946. Material collected at McGuire General Hospital, Richmond, Virginia.

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day was satisfactory and allowed comparison for follow-up studies, 35 patients had had open and 8 had closed head injuries and 7 had epilepsy; in 10, the examination was performed for other causes. In other words, a history of head injury was present in about two thirds of the cases. The interval between injury and encephalography, which varied from case to case, was over five months in approximately two thirds of the cases.

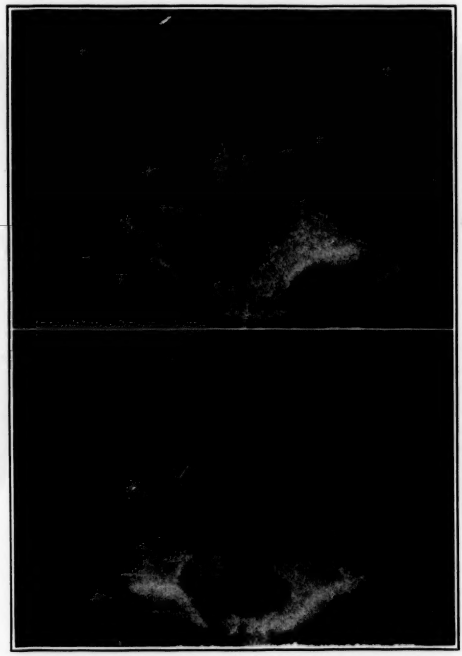

FIGURE 2. *Case 1.*

The lower film, an anteroposterior view taken on the second day, shows enlargement of both lateral ventricles, compared with almost normal-sized ventricles on the first day. There was a history of trauma four times in eight years, all of them due to automobile accidents, the last having occurred five months previously. There had been rapid mental deterioration for four months. Two hundred and forty cubic centimeters of air was injected. Clinically the patient was suspected of having brain atrophy, and the appearance of the ventricles on the second day corresponded with the clinical picture.

Methods of Examination

The patient was usually prepared with 10 mg. of morphine and 0.4 mg. of scopolamine. The examination itself was performed under Pentothal anesthesia, 30 to 45 cc. of a 2.5 per cent solution being administered intravenously. The patient started to become active toward the end of, or immediately after, the completion of the examination. Two needles were introduced into the lumbar canal in two adjacent interspaces. About 20 cc. of fluid was removed and replaced by the same amount of air. This procedure was repeated until a dry tap was obtained. The total amount of fluid removed varied, of course, from case to case. Its importance in relation to the second-day enlargement of the ventricles is discussed below.

Routine roentgenologic examination was done with the patient in the horizontal position and the

central ray in the vertical position. Anteroposterior, posteroanterior and lateral stereoscopic views represented the routine examination; additional films were taken in individual cases. The tube-film distance was held constantly at 90 cm.; the projection in various positions was standardized as much as possible. The first examination was made in the early hours of the afternoon. The second-day examination was usually done sometime during the forenoon, so that the actual time elapsed between the two examinations was between eighteen and twenty-two hours.

Various methods of measuring ventricles have been devised.[1, 8, 9] We used two different procedures. On the basis of past experience, the size was first judged without measurement. The ventricles were classified as normal, moderately enlarged and enlarged and were then measured. Whenever possible, the following separate measurements were made on the anteroposterior projection with the patient in the horizontal position: the distance between the superior medial corner of the ventricle and the convex lateral border of the ventricular body (Fig. 1*a*); the distance between the superior medial corner and the superolateral corner of the ventricle (Fig. 1*b*); and the distance between the superior medial corner of the ventricle and the concave lateral wall of the anterior horns (Fig. 1*c*).

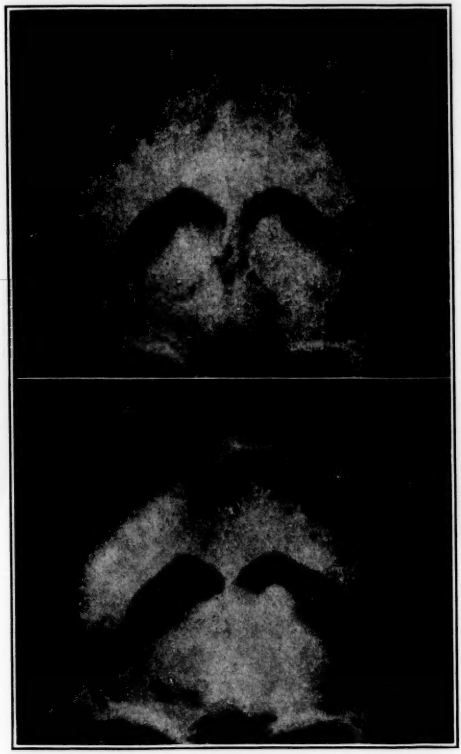

FIGURE 3. *Case 1.*
These films are first-day (upper) and second-day (lower) posteroanterior views.

The last line formed an angle of approximately 30° with the horizontal; it was at times difficult to measure and, if so, was quite arbitrary.

Like Paul and Erickson,[3] we found the first measurement to be of particular significance for the actual size of the ventricles, whereas the others were (Fig. 1*a*) was 1.1 cm. or less as normal, 1.2 cm. to 1.4 cm. as usually normal, 1.5 cm. as borderline, and 1.6 cm. as dilated. These standards were of course somewhat arbitrary, but for comparative studies they were found to be quite satisfactory. The influence of a slight variation in projection on the size

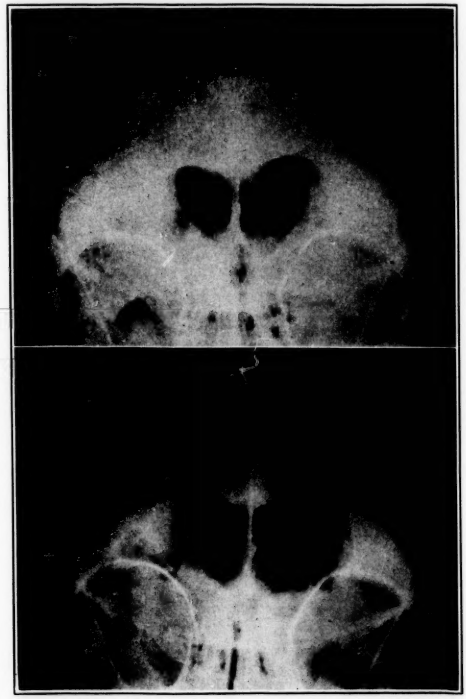

FIGURE 4. *Case 2.*

These films show increased ventricular enlargement two days following encephalography (no films were taken on the first day following the first examination). There was no history of injury. The patient had experienced disorientation and mental confusion for six weeks. There had been slight transient papilledema. One hundred and forty cubic centimeters of air was injected. The diagnosis was brain atrophy.

at times helpful in cases of localized loss of brain substance. The decision regarding the normal limit of the size of the ventricle is of necessity arbitrary in borderline cases, in spite of studies made in large groups of probably normal encephalograms.[1, 8] After a time, our estimated enlargement and that shown by actual measurement coincided fairly well, although in borderline cases there were some differences. We considered a ventricle whose width of the ventricles was checked in a number of cases. It was found to have a marked influence on the "height"[10] of the ventricles but produced only negligible differences in the measurements used.

RESULTS

Size of Ventricles

Of the 60 cases in which comparative second-day measurements were possible, the ventricles were

found to be larger on the second day in 23 (38 per cent). The enlargement was usually seen in the anteroposterior, posteroanterior and lateral projections, but it was most easily recognizable in the anteroposterior projection (Figs. 2, 3, 4, 5 and 6). In 18 cases, the second-day enlargement was considered to be slight, whereas in 5 a marked increase in the size of the ventricles occurred in the twenty-four-hour period. A slight decrease was seen in 3 cases. In 12 cases, there was no appreciable change in the size of the ventricles on the second day, and in 22 the amount of air in the ventricles on the second day was not sufficient for measurement. In other words, slightly more than a third of the total group of cases showed some enlargement of the ventricles, on the second day, whereas approximately 8 per cent of the total group showed marked enlargement. Of 43 patients who had suffered closed or open head injuries, 18 showed some enlargement. All the cases with pronounced second-day enlargement fell into the group with head injuries. In brief, of the patients with head injuries, approximately 42 per cent showed some second-day enlargement of the ventricles, and in 12 per cent of these the enlargement was marked.

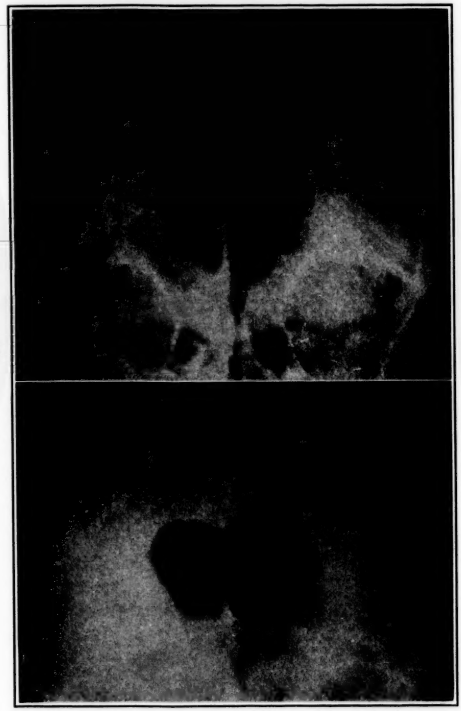

FIGURE 5. *Case 3.*

These films, which are anteroposterior views, show obvious second-day increase in ventricular enlargement. The patient had been struck on the head by a baseball six years before and was unconscious for eight days. He had had generalized convulsions for the previous five years. Neurologic examination was negative. Two hundred and ten cubic centimeters of air was injected.

It is interesting to compare our findings with those of Paul and Erickson.[3] Our technic was slightly different from theirs, inasmuch as our studies

were made with the patient in a horizontal and not in a vertical position. Only a few of our cases showed any reduction in the size of the ventricles, and the degree of decrease was not striking. It is likely that in many of our cases in which measurement in the horizontal position was not possible on the second day the ventricles would have been decreased in the upright position. The smaller percentage of encephalographic material. Our impression is that its incidence will be fairly high in any group of cases with cerebral atrophy.

Information about the amount of spinal fluid drained at the time of encephalography was available in 36 cases of the group. In 26 cases, less than 150 cc. had been drained. Second-day enlargement was slight in 6 and marked in 3. Of 10 cases in which

FIGURE 6. *Case 3.*
These films are left lateral views on the first and the second day.

second-day enlargement noticed in the material presented by Paul and Erickson[3] may be partially attributed to the difference in the position of the patient, but was more probably due to the fact that only 18 per cent of their cases were post-traumatic, as compared with 71 per cent in our series. In this connection, it is noteworthy that of the 7 cases in which Paul and Erickson found second-day enlargement, there had been trauma in 4. Thus, second-day enlargement is obviously more frequent in post-traumatic patients than in the routine average more than 150 cc. had been drained, 4 patients showed slight and 2 marked second-day enlargement. In other words, the incidence of second-day enlargement was slightly higher if more than 150 cc. of cerebrospinal fluid had been obtained.

In addition to the striking increase in the size of the ventricles, abnormalities observed in some of the cases made the second-day study worth while. The importance of the demonstration of porencephalic cysts has been emphasized by Pendergrass and Hodes,[4] as well as by Paul and Erickson.[3] In 3

of our cases the cyst was visible only on the follow-up film, whereas in 4 others the cyst, although visible at the original examination, was better filled on the second-day examination (Figs. 7 and 8). In addition to the better demonstration of porencephalic cysts, second-day examination is considered of value in cases in which only one ventricle was filled on the first day and in which, by proper posturing of the head, filling of the other ventricle was obtained on the second-day examination.[3, 4]

In 8 of our cases a bulging of the interventricular septum was noted on the second-day roentgenograms (Fig. 9). This was either not present or much less pronounced on the original films. The change in the appearance of the septum is difficult to explain. At times it was purely an optical phenomenon, since different parts of the wavy septum were demonstrated on different days of the examination. At other times, however, the septum actually seemed to bulge markedly on the second day, whereas it had been absolutely straight on the first day. It is likely that this is evidence of a change in the shape of the ventricles from day to day, the ventricles being not so fixed in size and shape as they are usually assumed to be. Septal bulging was found only in patients with dilated ventricles, and it is likely that the shape of the ventricles is particularly variable in patients with brain atrophy. In

FIGURE 7. *Case 4.*

The lower film, taken six days after the first examination, shows filling of a porencephalic cyst (no intermediate examinations were made). Note the defect of the frontal bone and the displaced bone fragments deep inside the vault. The patient had had a right frontal injury by a shell fragment two months previously, with immediate débridement. There were loss of memory and poor cerebrations. One hundred and forty cubic centimeters of air was injected.

7 cases the septum bulged toward the right side. There was no obvious relation between the side of maximum brain loss and the direction of the bulge.

Discussion

The increase in the size of the ventricles in some of our cases was quite unexpected. Although the follow-up examination, this was not always accomplished. A slight change in projection, however, caused no appreciable enlargement of the width of the ventricles, as we proved in especially conducted studies. In addition, a definite increase in the size of the ventricles on the second-day examination was found in cases in which the projections on the two

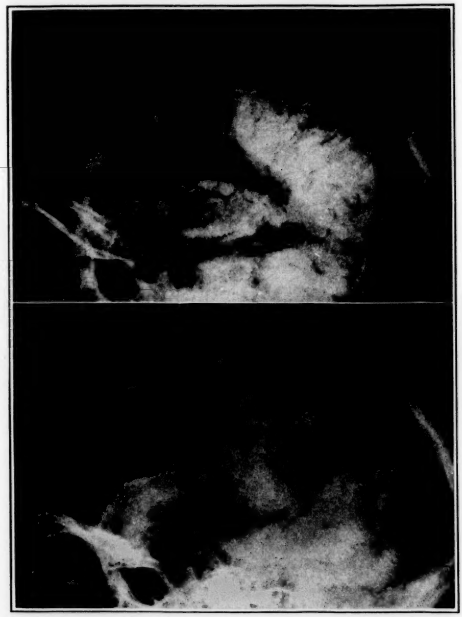

Figure 8. *Case 5.*

The lower film demonstrates second-day filling of a porencephalic cyst. Note the air-filled cyst above the dilated anterior horn. The patient had had a left frontoparietal injury due to grenade fragments and followed by débridement. There had been marked aphasia, which had improved. There was an old papilledema, with diminished vision, as well as slight headache and dizziness. The brain protruded through the skull defect, and most of the porencephalic cyst was within the protrusion. One hundred and eighty cubic centimeters of air was injected.

observation appears to be irrefutable, the explanation is difficult and debatable. An explanation on a purely optical basis can be excluded. The various examinations were performed under identical circumstances, and the same optical conditions existed. The size of the frontal sinuses was measured routinely and was found to be identical in the different examinations of a given case. Although an attempt was made to reproduce the original projection in the examinations were identical. In short, there can be no doubt that the ventricles in these cases were larger on the second day.

The question arises regarding the correct size of the ventricles in such cases. Were the ventricles on the first-day examination smaller than they normally are, or was there a transient abnormal increase in their size on the day following the introduction of air? Paul and Erickson[3] apparently as-

sume that the second-day dilatation is the artificial finding and give the following possible explanations. Gas, trapped at the base of the skull — in the cervical region, the cisterna magna or the subarachnoid space of the posterior fossa or even in the third or fourth ventricle — passed upward. This explanation appeared to be applicable to some of our cases. Rapidly emptying gas from the subarachnoidal space caused dilatation of the ventricles. An abnormality was present in the gaseous interchange between the air in the ventricles and the gases in the cerebral tissues or blood vessels. Finally, a difference in the intracranial pressure for the two days or, to a lesser extent, in the temperature altered the gas volume.

In our opinion, it is difficult to say whether the size on the first or the second day represents the normal condition of the ventricles. It is well known that the brain responds rapidly to decreased intracranial pressure by an increase in volume. Such increase may be due to either edema or hyperemia of the brain. One may assume that the sudden withdrawal of all the ventricular fluid represents a marked trauma to the brain, which quickly responds to such trauma. In other words, the enlarged brain presses from all sides on the ventricles and decreases their size. Within twenty-four hours the irritation caused by the sudden withdrawal of fluid subsides and the ventricles resume their normal size. In favor of this assumption is the fact that in patients in whom films were taken after twenty-four hours, no further increase in the size of the ventricles was noted. This theory also explains why, in a few cases of severe, repeated brain trauma with clinical signs of considerable brain damage, the ventricles

FIGURE 9. *Case 6.*

The lower film shows the second-day bulge of the septum pellucidum. The patient had had grand-mal attacks for four years, but there was no history of trauma.

appeared normal, or almost normal, on the first day and showed dilatation on the second day. This second-day dilatation of the ventricles coincided better with what had been expected from the clinical picture.

On the other hand, it is conceivable that the first-day examination represented the normal size of the ventricles and that the irritation of the air in the ventricles caused hypersecretion to such a degree that it was inadequately drained through the foramens of the fourth ventricle and thus led to dilatation of the ventricles. In favor of this theory, is the fact that the second-day enlargement was generally seen if large amounts of air had been introduced. The evidence against this theory, however, is stronger than that in favor of it. In some cases the ventricles not only were larger on the second day but also contained more air than on the first day. Hypersecretion, with an increase of the pressure within the ventricles, should press the air out into the subarachnoid space and should not attract it.

Measurements of the cerebrospinal-fluid pressure at the time of introduction of air and again on the day following the encephalography would be interesting. The latter measurements were not made in this series.

Although a satisfactory explanation of second-day enlargement is not possible, we are inclined to believe that if the ventricles enlarge within twenty-four hours, the second-day examination represents the normal or resting size. Further clinical and experimental studies regarding this problem appear to be necessary.

There are certain practical applications of the recognition of this phenomenon. We have not seen pronounced second-day enlargement in cases without fairly marked clinical evidence of brain damage. For this reason, routine study of the ventricles twenty-four hours after the introduction of air is advised. This study is particularly indicated in cases with old head injuries or in those in which atrophy from other causes is suspected. The second-day examination not only often shows bilateral or unilateral dilatation that was not present on the first day but also, at times, emphasizes and clarifies certain anomalies that were equivocal on the first day. We have seen a few cases in which the second-day examination clearly showed the presence of brain atrophy, whereas that on the first day had left the question of tumor or atrophy open.

Summary

Observations in 60 patients who had roentgenologic studies on the day following routine encephalography are described. Thirty-five of these had open and 8 had closed injuries; 7 were epileptic, and in 10 the examination was performed for other reasons.

A slight decrease in the size of the lateral ventricles on the second day was seen in 3 cases. The lateral ventricles were slightly larger on the second day in 18 cases, and there was marked enlargement in 5 cases. Each of the last cases had a history of preceding head injury.

At times, a peculiar bulging of the interventricular septum was seen on the second day. Second-day studies showed filling of porencephalic cysts that were not filled, or incompletely filled, on the first day.

The mechanism of second-day enlargement is discussed.

Roentgenologic studies on the day following encephalography appear to be indicated, particularly when the examination is performed for the diagnosis of post-traumatic conditions.

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ARTHRITIS IN THE MEDITERRANEAN THEATER OF OPERATIONS*

II. Clinical Description of Hypertrophic Arthritis, Arthralgia and Psychogenic Rheumatism

LIEUTENANT COLONEL CHARLES L. SHORT, M.C., A.U.S.

THE types of joint disease often encountered in the Mediterranean Theater, in addition to rheumatoid arthritis, which was discussed in a previous paper,[1] included hypertrophic arthritis, arthralgia and similar disorders, and psychogenic rheumatism.

HYPERTROPHIC ARTHRITIS

The aspects of hypertrophic arthritis that seemed of importance in the Theater are covered in this section, no attempt being made to discuss the definitive treatment of the disease. Because it was usually employed, the designation "hypertrophic arthritis" is used throughout to avoid confusion, although in some ways its synonyms, such as "degenerative joint disease" and "osteoarthritis," are preferable. When possible, the two types of hypertrophic arthritis encountered in military as well as in civilian practice are differentiated and compared. These are the primary usually more generalized type and the secondary more localized type, resulting from structural abnormality, previous trauma or joint damage caused by another form of arthritis.

Incidence

The exact incidence of hypertrophic arthritis in the Theater could not be determined. A total of 621 cases were reported by questionnaires from ten general hospitals. This figure may be somewhat higher than the actual one because of both duplication of cases and a tendency to diagnose the disease from x-ray findings when it might not be responsible for the patient's disability. Hypertrophic arthritis made up about 25 per cent of the total number of patients with arthritis admitted to general hospitals, a ratio approximating that found by Boland and Corr[2] in a general hospital in the United States. The prevalence of this disease, which is generally regarded as an affection of middle or old age, in an overseas theater seems unusual until the large number of soldiers in their late thirties are taken into account, as well as the still older commissioned and noncommissioned officers of the higher grades. In addition, the secondary type, which is usually symptomatic at an earlier age, made up over a third of the cases diagnosed as hypertrophic arthritis.

Etiology

Table 1 presents the age distribution of 51 cases of hypertrophic arthritis, with a comparison between primary and secondary types. The mean age of the primary type, forty-one years and eight months, surpassed by about ten years that found for a series of patients with rheumatoid arthritis.[1] In spite of the small number of cases, a significant difference also seemed to exist between the two forms of hypertrophic arthritis, since the majority of cases in the primary type occurred in the fifth decade of life in comparison with the fourth decade in most of the other group.

The cause of the primary type of hypertrophic arthritis remains unknown, although aging and degeneration of articular cartilage certainly constitute the underlying process. Other etiologic factors are therefore difficult to assess, although exposure to cold and dampness, sleeping on the ground and the strain of long marches, especially in mountainous country, and of riding in army vehicles were all cited by patients as precipitating causes of disabling symptoms. Acute trauma to a joint in the form of a strain or a direct blow from a fall also preceded the onset in certain cases or caused an acute exacerbation in a previously mild process. All these factors were also operative in bringing patients with the secondary type to medical attention, although the underlying process was usually initiated years previously. Table 2 lists a classification of the factors apparently causing or accelerating the development of the secondary type of hypertrophic arthritis. These data were derived from the records of 100 such patients in a combined series of 270 cases in which the diagnosis was made. A study of the table reveals that trauma was a cause about three times as frequently as a long-standing or congenital structural abnormality. The types of trauma were about equally divided between an actual fracture, either ununited or healing in a position to throw strain on one or more joints, and direct or indirect trauma to the joint itself. One patient developed hypertrophic arthritis of the shoulder joint secondary to the continued presence in the joint of shot-gun pellets, acquired four years previously. This case, together with others previously reported, points to the importance of the removal of missiles or shell fragments from joints to prevent what has been called "foreign-body arthritis."

Diagnosis and Clinical Features

From the data given above regarding age incidence and from the general experience in the Theater, hypertrophic arthritis was naturally suspected when an older patient presented himself with joint complaints. A previous history of similar symptoms, usually low-grade, was frequently obtained, especially in response to unfavorable climatic conditions or unusual exertion. In many cases there

*The following material represents a condensation and a certain amount of revision of parts of a clinical monograph on arthritis prepared at the direction of the Surgeon, Mediterranean Theater of Operations, United States Army, and submitted to him in final form in June, 1945.

had been remissions, either spontaneous or in response to rest or hospitalization. In the secondary type, as might have been expected, the underlying cause was usually operative and productive of some symptoms before entrance on active duty.

In common with civilian experience, objective joint changes were less striking in hypertrophic than in rheumatoid arthritis, acute synovitis or effusion being rare and usually occurring in response to superimposed recent trauma. Findings were limited largely to crepitus, tenderness, bony irregularity and slight degrees of limitation of motion. The distribution of the arthritis was of some assistance in diagnosis and corresponded to that noted in civilian life. In the 33 patients with the primary type listed in Table 1, the upper extremities, including the shoulders, fingers and acromioclavicular joints, were rarely the seat of symptoms. Heberden's nodes, except for the form secondary to athletic or occupational trauma, were seldom noted in soldiers in the Theater. The spine was oftenest involved, chiefly in the lumbar region, although radicular pain associated with hypertrophic arthritis of the cervical spine was occasionally recorded. Next to the spine, joints of the lower extremities were most frequently affected, especially the hips and knees. In one hospital, a number of patients with hypertrophic changes in the tarsal joints had been admitted with a diagnosis of foot strain.

In the primary type of hypertrophic arthritis, bilateral involvement of corresponding joints was present in a fair number of cases, but a symmetrical distribution was nowhere near so striking as in rheumatoid arthritis. Bilateral arthritis was unusual in the secondary form, in which there was a wider distribution of articular involvement (Table 2) and in which the spine, knees and hips were generally involved.

Laboratory tests were of little assistance in the diagnosis of either type of hypertrophic arthritis. The sedimentation rate was usually normal. Roentgenograms, on the other hand, were essential in the diagnosis. In fact, medical officers without exception stated that a definite diagnosis of hypertrophic arthritis was not made in the absence of x-ray changes. As pointed out below under arthralgia, however, the presence of an early hypertrophic arthritis without x-ray changes may have been surmised but not diagnosed in certain patients on account of their age and the localization of symptoms.

Since the diagnosis of hypertrophic arthritis depended almost entirely on positive x-ray findings, there is little need of discussing the differential diagnosis. The danger of assuming that the patient's symptoms were necessarily associated with these findings was apparent, however, especially in dealing with radicular pain, in which the ruling out of other possible causes was rightly considered a prerequisite to a final diagnosis of hypertrophic arthritis alone. In a certain number of cases the symptoms were chiefly or entirely dependent on neurosis rather than the arthritis demonstrated by x-ray examination. The finding of mild degrees of hypertrophic arthritis in older soldiers, who may have had skeletal symptoms but whose chief trouble was a lack of sufficient stamina for combat duty, led to the use of this diagnosis as a convenient method of assigning the patients to limited service. Among 132 patients sent to limited service with a diagnosis of arthritis by the Third Convalescent Hospital, a report states that 20 should more properly have been designated "over age for combat service."

TABLE 1. *Age Distribution of Patients* with Various Types of Arthritis, as Obtained from the Records of the Twelfth General Hospital.*

AGE	PRIMARY HYPERTROPHIC ARTHRITIS		SECONDARY HYPERTROPHIC ARTHRITIS		ARTHRALGIA		PSYCHOGENIC RHEUMATISM	
	NO.	PERCENTAGE	NO.	PERCENTAGE	NO.	PERCENTAGE	NO.	PERCENTAGE
yr.								
15-19	0	0.0	0	0.0	1	1.2	2	3.9
20-24	0	0.0	2	11.8	2	2.3	9	17.6
25-29	1	3.2	2	11.8	15	17.4	15	29.4
30-34	4	11.8	8	47.0	17	19.7	17	33.4
35-39	5	14.7	4	23.5	26	30.4	7	13.7
40-44	11	32.2	0	0.0	17	19.7	1	2.0
45-49	11	32.2	0	0.0	7	8.1	0	0.0
50-54	2	5.9	1	5.9	1	1.2	0	0.0
Totals	34		17		86		51	

*The mean age was 41.7 years among patients with primary and 33.3 years among those with secondary hypertrophic arthritis, 34.1 years among those with arthralgia, and 29.2 years among those with psychogenic rheumatism.

Treatment and Course

Since articular cartilage is a tissue displaying little capacity for repair, the changes in hypertrophic arthritis are irreversible, and the condition is usually slowly progressive. No specific medical treatment is available, and surgery to correct causative or accelerating factors was rarely indicated in an overseas theater. The measures employed were therefore chiefly symptomatic and consisted largely in rest and freedom from weight bearing, as well as in the application of heat. Certain cases were bene-

fited by orthopedic measures, chiefly exercises to improve bodily mechanics and strengthen supporting muscles, with the occasional use of foot plates or supports for the lower back. Except in severe cases, a trial of such methods seemed justified with a view to returning the patient to some form of duty, usually at a lower level of physical activity than in his previous assignment. In planning the extent of the treatment and hospitalization, it was of assistance to realize that the disease is prone to partial or even nearly complete symptomatic remissions, both spontaneous and in response to the removal of environmental or occupational factors causing excessive strain on the affected joints. Each case was necessarily handled individually, with the knowledge that the severity of the arthritis, as evidenced by the x-ray film, did not necessarily foretell the outcome.

Disposition

Combined figures for the disposition of 621 patients with hypertrophic arthritis obtained from ten general hospitals were as follows: to duty, 66 (10.6 per cent); to limited service, 170 (27.4 per cent); and for evacuation, 385 (62.0 per cent). In addition, the probability exists that in many diagnosed as hypertrophic arthritis in station and evacuation hospitals and in the Third Convalescent Hospital the patients were returned to some form of duty without having reached a general hospital.

The disposition in each case was often unpredictable when the patient was first admitted. Cases in which the indications for evacuation were clear from the start included severe cases of the primary type and those with the secondary type in which the underlying cause made improvement under ordinary measures unlikely, as well as patients who had been tried unsuccessfully at limited service and those whose symptoms failed to improve sufficiently with previous hospitalization. It seemed fully justifiable to classify the remainder for limited service, with the expectation that, in the majority, the symptoms would be sufficiently controlled to enable the patients to be of use in an assignment in the Zone of Communications not involving undue strain on the involved joints. A small remainder, with mild symptoms responding readily to treatment or with light assignments, were sent to full duty. Finally, the impression was gained that a smaller number of patients were evacuated in the Italian than in the North African phase of operations.

Arthralgia, Myositis and Myalgia

This group might be called the "unwanted stepchild" of the arthritic diseases as seen in the Theater. The patients presented complaints without objective findings, were often suspected of neurosis or malingering and furnished real difficulty in diagnosis, treatment and disposition. But, since the cases made up the largest proportion with complaints referable to the joints and other supporting structures, the condition had to be dealt with, if not always to the satisfaction of the medical officer's scientific principles, at least in a practical manner. In the collection of data, even more than the usual confusion associated with the nomenclature of the arthritic diseases was encountered. The most frequent term employed in the diagnosis of patients with pain and stiffness of the joints without evidence of a real arthritis or an underlying neurosis was "arthralgia." Myositis or myalgia was employed

Table 2. *Classification of the Underlying Causes of Secondary Hypertrophic Arthritis in 100 Patients.*

Cause			No. of Cases
Structural abnormality:			25
Spinal disease:		11	
Osteochondritis	5		
Congenital defect, fifth lumbar vertebra	4		
Scoliosis	1		
Spondylolisthesis	1		
Hip disease:		10	
Legg-Perthes disease	4		
Slipped epiphysis	3		
Congenital dislocation	1		
Coxa vara	1		
Os acetabuli	1		
Obesity		3	
Genu varum		1	
Previous trauma:			74
Old joint injury:		33	
Knee	15		
Spine	10		
Elbow	2		
Shoulder	2		
Ankle	1		
Hip	1		
Metatarsophalangeal	1		
Acromioclavicular	1		
Old fracture:		41	
Spine	8		
Ankle	8		
Femur	4		
Wrist	4		
Elbow	7		
Knee	2		
Humerus	2		
Tarsus	2		
Patella	1		
Hip	1		
Pelvis	1		
Acetabulum	1		
Previous arthritis:			1
Infectious, hip	1		

when the symptoms were localized to the region of muscles rather than joints and when the process was relatively acute. A few hospitals used "fibrositis" or the variation "fibromyositis," according to British usage, which is followed by some in the United States under the assumption that a definite pathologic process is responsible — namely, inflammation of white fibrous tissue. Since this assumption has never been adequately proved, it seemed preferable to discard the term "fibrositis" for the present purpose. Furthermore, since a definite myositis is as incapable of proof as fibrositis in most cases and since, in many cases, the symptoms were in the region of both joints and muscles, it was decided to utilize in this section the terms "arthralgia" and "arthralgia group." In this way, unwarranted implications concerning pathology and pathogenesis were avoided, as well as those regard-

ing the presence of a chronic or progressive disease. The objections noted above apply also to the use of the diagnosis "chronic arthritis, type undetermined," as employed in some hospital records. Since no attempt was made to cover the subject of organic or functional disorders of the lower back, unless caused by some form of arthritis, patients with pain and stiffness confined to this region were not included under the heading of arthralgia. More definite and localized affections are also not considered, such as strains, sprains, bursitis and tenosynovitis.

Incidence

No exact figure can be given for the incidence of arthralgia in the Theater. A total of 918 cases were reported by questionnaire from ten general hospitals, 497 being labeled arthralgia and 421 myositis or myalgia. This figure probably needs little correction for duplication of cases and, in fact, should be higher, with the addition of patients from the group with the diagnosis of chronic arthritis, type unspecified. The patients with arthralgia probably made up nearly 40 per cent of the total number with arthritis admitted to general hospitals, as compared with about 25 per cent each for rheumatoid and hypertrophic arthritis and 10 per cent or less for specific infectious arthritis. The incidence of arthralgia was probably higher in lower medical echelons in the Theater, since many patients were undoubtedly discharged to duty without having been admitted to a general hospital. Patients without objective findings of joint disease were reported to make up the majority of those admitted with articular complaints to one evacuation hospital, whereas they comprised at least half of those listed under arthritis in the records of the Seventy-third Station Hospital. Other available figures in World War II include an incidence of 70 per cent for "fibrositis" among the patients with arthritis admitted to two British general hospitals in 1940,[3] contrasted with 5 per cent in a general hospital in the United States[2] and 13.6 per cent at the Army and Navy General Hospital, Hot Springs, Arkansas.[4] The smallness of the last percentages may have been due to the fact that in the American hospitals a special search was made for cases of psychogenic rheumatism among the patients without evidence of real arthritis. In World War I, in about 25 per cent of the 65,192 patients admitted to Army hospitals for arthritis the diagnosis was muscular rheumatism or myositis.[5]

Etiology

A sample to show the age distribution of patients with arthralgia was obtained from a series of 86 patients in the records of the Medical Service of the Twelfth General Hospital (Table 1). Patients were selected, irrespective of the hospital diagnoses, who complained of pain and stiffness in and around the joints without clinical, laboratory or x-ray evidence of arthritis. This group tended to be made up of older patients, the mean age (thirty-four years and one month) being higher than that in rheumatoid arthritis (thirty years and nine months), but considerably lower than that in hypertrophic arthritis (forty-one years and eight months). This finding, although noted in other hospitals, may be only apparent, since a frequent practice in the Theater was to return young patients with arthralgia to duty, whereas older soldiers were likelier to be sent to a general hospital to be classified for limited service. No clue was obtained in the Theater regarding the actual causes of arthralgia, but precipitating factors included severe exertion and exposure to cold and dampness. An increase in the number of cases from the Fifth Army hospitals was noted at the Third Convalescent Hospital at the beginning of the cold and rainy season. There were few cases of acute infections of the upper respiratory or gastrointestinal tract directly preceding the onset of symptoms, although such conditions were often accompanied by transitory skeletal aches and pains.

Diagnosis and Clinical Features

In the diagnosis of arthralgia, a careful history was all important. Unless the patient's symptoms were characteristically "rheumatic," a strong suspicion of psychogenic rheumatism properly arose. The necessary points for a rheumatic symptomatology may be listed as follows: morning stiffness and an increase in pain after sitting or lying in one position during the day; aggravation by cold, dampness or weather changes; and at least temporary relief from heat, moderate exercises and aspirin. Severe exertion, with overuse of the joints and muscles during the day, may, of course, cause an increase in pain or stiffness toward evening as well as on arising. It was found necessary for the examiner to avoid the use of leading questions and to realize that a soldier may have had an opportunity to compare notes with a fellow sufferer from arthritis, especially on a hospital ward. Marked constitutional or vasomotor symptoms were unusual in this group and, when present, suggested the possibility of a mild rheumatoid arthritis. The same observation held true of neurologic symptoms, with motor weakness and tremor again suggesting rheumatoid arthritis. Bizarre paresthesias or numbness in contrast to genuine pain and stiffness were often indicative of a psychogenic origin of symptoms.

The physical examination in these patients showed no objective changes in the form of swelling or joint effusion, although the patient might complain that his joints felt swollen. Mild or moderate degrees of limitation of motion, which were occasionally present, were associated with muscle spasm or stiffness rather than actual loss of joint function. Tenderness over joints, muscles and other supporting structures and pain on motion of joints or on stretching of muscles were frequently elicited. The

distribution of involvement is presented in Table 3. As indicated by the figures, many patients had involvement of multiple regions, the lower extremities being affected about twice as often as either the back or the upper extremities.

The sedimentation rate was uniformly normal in patients with arthralgia. The one exception consisted in a few patients with marked tenderness, chiefly localized to the lumbar paravertebral muscles, in all of whom the sedimentation rate was moderately elevated, and in some cases there was a suggestion of muscle swelling, along with fever and leukocytosis. The course was that of slow improvement, with a disappearance of symptoms and signs and a return to normal of laboratory findings. Despite careful observation over a relatively long period, they never developed evidence of disease of bones or joints and were eventually returned to duty. In such patients alone, a diagnosis of myositis seemed justified. X-ray examination of the involved region was performed in nearly all hospitalized patients with arthralgia, with negative results.

Differential Diagnosis

A final diagnosis of arthralgia in a patient with the findings described above depended on the exclusion of either a real arthritis or an underlying neurosis as a cause for symptoms. Much difficulty arose in the elimination of an early, mild rheumatoid arthritis, since the articular distribution, including symmetry, often suggested this disease. The differentiation of hypertrophic arthritis depended almost entirely on the x-ray findings. In many patients, on account of the age distribution (Table 1) and the localization of symptoms to the back and lower extremities (Table 3), the question of an early hypertrophic arthritis, without x-ray changes, was properly raised. In such cases the practice was to employ arthralgia as an immediate diagnosis, with the reservation that hypertrophic arthritis might be revealed by x-ray study at a later date. A careful orthopedic check-up examination proved useful in a number of patients by revealing structural abnormalities that might previously have been overlooked. These included foot strain, genu valgum, varum or recurvatum, bursitis in various localities and organic changes of the spine. Rheumatic fever was rarely difficult to distinguish, but electrocardiographic study of an occasional case showed a definite increase in the PR interval and favored a presumptive diagnosis of this disease.

Most important of all to eliminate in the arthralgia group was a neurotic basis for symptoms, or so-called "psychogenic rheumatism." The distinction was made on two grounds: the demonstration of an underlying neurosis, with psychiatric aid in certain cases; and the finding, after careful evaluation, that the patient's symptoms were not characteristically "rheumatic," as described above. In certain patients, in whom both neurosis and arthralgia were apparently present, careful clinical judgment was required to ascertain the predominant factor, the use of two diagnoses occasionally being justified. The medical officer was helped in this decision by the age of the patient (the symptoms seemed genuine more frequently in older patients) and, if available, by the opinion of the soldier's commanding officer. It seemed probable, however, that many cases of neurosis had inaccurately been labeled arthralgia or a similar diagnosis. The much larger number of patients found in a general hospital in the United States with psychogenic rheumatism than fibrositis has been mentioned above,[2] whereas in civilian practice in England, Halliday[6] considered the disability due to psychogenic factors in 37 per cent of 62 insured patients with a diagnosis of rheumatism. The question of malingering occasionally arose in these patients but seemed incapable of proof. Conscious or unconscious exaggeration of symptoms was not unusual, however, and was generally readily recognized and appropriately handled by the medical officer.

TABLE 3. *Areas of Body Affected by Arthralgia among 86 Patients, as Obtained from the Records of the Twelfth General Hospital.*

AREA		NO. OF CASES
Upper extremities:		62 (31%)
Shoulders	33	
Elbows	10	
Hands	8	
Wrists	11	
Back:		49 (24%)
Cervicodorsal	15	
Lumbosacral	34	
Lower extremities:		90 (45%)
Knees	46	
Hips	19	
Legs	12	
Ankles	11	
Feet	2	

Treatment and Course

The treatment of the patients with arthralgia was usually symptomatic and included the benefits of hospital shelter and care (rest, warmth, sleeping off the ground and avoidance of severe exertion), relief of pain with salicylates and physical therapy in the form of heat, massage and exercises. Some success was attained in the more acute, localized types, but the generalized forms seemed to derive only temporary, if any, alleviation of symptoms. In the latter, prolonged hospitalization proved ineffective and indeed wasteful of bed space and manpower once the diagnosis had been established. A decision regarding diagnosis and disposition was therefore indicated as quickly as possible. Along these lines, greater stress should have been laid on handling the problem more frequently in the Army area or in station hospitals in the Zone of Communications. In this way, with a relatively short period of observation, a sorting could have been made of cases

that were obviously functional, those with arthralgia and those with probably real arthritis. Fewer patients in the first two groups would then have needed to be sent to general hospitals.

Disposition

Figures from ten general hospitals showed the following proportion of dispositions for 918 patients with arthralgia: to duty, 538 (58.6 per cent); to limited service, 263 (28.6 per cent); and for evacuation, 117 (13.8 per cent). The percentage of patients with myalgia or myositis who were evacuated was essentially the same as that in cases of arthralgia, but a greater proportion of the former were returned to full duty. It was the opinion of medical officers questioned in evacuation and station and most general hospitals that a large majority of the patients with arthralgia were sent to duty. The exceptions comprised patients who had repeatedly failed at full duty, older soldiers who were otherwise unsuited for combat and those whose commanding officers believed that they should be reclassified. The small number evacuated were mainly soldiers who were unable to perform even limited service or those with a severe psychoneurosis in addition. The remainder were sent to limited service and were usually able to carry on without rehospitalization. In summary, from the general hospital figures, nearly 90 per cent of patients with arthralgia were returned to some form of duty, with the probability that an even larger proportion from evacuation and station hospitals were sent to full duty, rather than to limited service.

Psychogenic Rheumatism

The term "psychogenic rheumatism" was adapted from the work of Boland and Corr[2] at the Hoff General Hospital, Santa Barbara, California. Their definition of this condition is as follows: "states in which symptoms such as pain, stiffness, subjective sense of swelling or limitation of motion in the muscles or joints are caused, intensified or perpetuated by mental influences." A similar name, "psychosomatic rheumatism," was used in England for a number of years by Halliday,[7] who stressed the high incidence of this cause of articular symptoms, especially in cases labeled fibrositis. Although psychogenic rheumatism is a convenient and concise descriptive term for this group of patients, it should be made clear that it is not advocated as a final diagnosis in the records of Army or civilian hospitals. The usual diagnoses of neurosis or psychoneurosis, with the type and manifestations, should be employed.

Incidence

About one sixth (16.5 per cent) of 309 patients with articular complaints admitted to the Medical Service of the Twelfth General Hospital were believed to be suffering from psychogenic rheumatism. This proportion, which was equaled by that of patients with hypertrophic arthritis and was exceeded by patients with arthralgia (33.4 per cent) and rheumatoid arthritis (24.8 per cent), outnumbered the remainder, chiefly patients with specific infectious arthritis (8.0 per cent). At the Army and Navy General Hospital, in 18.8 per cent of patients with rheumatic complaints the diagnosis was psychogenic rheumatism,[4] a somewhat greater proportion — 60 (26 per cent) of 231 patients with peripheral joint involvement — being so classified by Boland and Corr,[2] who were especially interested in and on the lookout for this condition. These authors, in fact, found psychogenic rheumatism the most frequent cause of disability encountered in their series, although the incidence was only slightly in excess of that of rheumatoid arthritis.

Another method of approach to the incidence of psychogenic rheumatism was an estimate of the proportion of cases of neurosis manifested by symptoms referred to the joints, muscles or other supporting structures. In a study of patients admitted to the Fifty-sixth Evacuation Hospital with diagnoses of organic diseases, 174 patients whose symptoms were actually on a functional basis were recorded and classified. The diagnosis of some form of arthritis on admission was made in only 13 (7.5 per cent), as compared with 51 patients, or 29 per cent, admitted with gastrointestinal disease, 25 patients, or 14 per cent, with upper respiratory symptoms, 19 patients, or 11 per cent, with backache and 14 patients, or 8 per cent, with headache. These figures were also confirmed by neuropsychiatric opinion, which put articular manifestations of the neuroses at the end of a list including, in order of frequency, backache, gastrointestinal symptoms, headache and cardiovascular symptoms. At the Six Hundred and First Clearing Company, where patients with neuroses were gathered directly from the Fifth Army and were nearly all considered "battle incurred," this symptom-complex was rarely encountered. Most of the patients manifested "free anxiety," and it was believed that somatic symptoms in this group usually developed after hospitalization. Major hysteria, however, in the form of gaits, postures and joint symptoms that might mimic a real arthritis, made up perhaps 2 or 3 per cent of the admissions. A few such patients were also seen in hospitals in the Zone of Communications, only 10 being listed in the questionnaires. The experience of Boland and Corr[2] in the United States is at variance with the information available for the Mediterranean Theater. These authors stated that disabling psychogenic complaints, as observed in an Army general hospital, were displayed frequently in the joints and muscles and were comparable to their occurrence in the gastrointestinal and cardiovascular systems. The difference may lie in the assiduity with which they searched for this type of

case and in the fact that they included patients with low-back symptoms on a functional basis.

Etiology

The immediate precipitating emotional cause, as Boland and Corr[2] have pointed out, was difficult to elicit and was not often found in the records. Factors common to the development of other manifestations of neurosis in troops overseas were undoubtedly active and need not be listed here. In general, the patients seemed younger than those with an actual arthritis and, as one medical officer put it, "can be picked out of the younger patients with arthralgia." Table 1 shows the age distribution in 51 patients from the records of the Twelfth General Hospital. A comparison with other types of arthritis in this table reveals that patients with psychogenic rheumatism approximately equaled in age those with rheumatoid arthritis, but comprised a definitely younger group than those with hypertrophic arthritis or arthralgia. In Boland and Corr's[2] series, on the contrary, no relation to age was found.

Except in the experience of the Six Hundred and First Clearing Company, precipitation or aggravation by combat was frequent, about half the patients admitted to the Twelfth General Hospital having given this history. A distinction was not always made in the record, however, between the onset of the anxiety state and the actual somatic fixation. In common with the findings of Boland and Corr,[2] about half the patients presented a history of articular symptoms. In many of these cases there was an actual history of injury to a joint or a preceding disease accompanied by pains in the joints, such as rheumatic fever, influenza or meningitis (stiff neck), but without objective changes on examination. In most cases it was impossible to determine from the record whether the patient had had psychogenic rheumatism in the past or whether the previous injury or disease had merely provided a site for somatic fixation of the neurosis. A family history of "rheumatism," found in over a third of the patients studied in the United States, was not prominent in the Theater, except for a few striking cases, but might have been brought to light oftener by direct questioning. The presence of an underlying neurosis, dating back to civilian life in over half the cases, evidently furnished the cause for the development of somatic symptoms. This was brought out by past and family histories of neuropathy and by the elicitation of other psychosomatic symptoms, some on a frankly hysterical basis. About half the patients at the Twelfth General Hospital gave evidence of general or localized functional symptoms in addition to those directed toward the joints. The role of hospitalization in the development of somatic symptoms, as postulated by the Six Hundred and First Clearing Company, was difficult to demonstrate with numerical data, but most medical officers questioned could recall specific cases, occasionally with direct contagion from fellow patients with joint complaints.

Diagnosis and Clinical Features

The diagnosis of psychogenic rheumatism depended on two criteria. The first was the ruling out of organic disease as the important factor in the patient's complaints, with the aid of the principles outlined in the sections on individual types of arthritis and, in doubtful cases, with the help of a careful orthopedic examination. The second consisted in the establishment of a positive diagnosis of neurosis, by a family or past history of neuropathy and by the presence of other psychosomatic symptoms. Persistence or return of symptoms localized to the site of previous disease or injury was useful in arousing suspicion, as well as a family history of joint disease with symptoms similar to those of the patient. Bizarre complaints, including paresthesias and numbness and symptoms not typically "rheumatic" (as outlined above), served to direct the examiner's attention to functional disease. In certain patients the opinion of a psychiatrist was of great value, either in confirmation of the internist's opinion or to bring out the patient's neurotic background by a trained psychiatric approach.

The physical examination in these patients was usually entirely negative. A certain number presented joint limitation or deformity, associated with muscle spasm. Others showed mild residual changes that were obviously not responsible for the symptoms. In a third group, areas of hysterical anesthesia were present, either in a glove and stocking distribution or in the neighborhood of the involved joints. Patients with hysterical gaits and postures, including camptocormia, and with joint deformity without actual arthritis were usually easily identified, sometimes with the aid of sodium amytal narcosis. The distribution of articular involvement corresponded to that found by Boland and Corr,[2] the majority complaining of symptoms in the back and lower extremities, compared with a smaller number of cases with symptoms in the upper extremities or generalized.

The psychiatric diagnosis made in the 51 patients from the Twelfth General Hospital was variable, without the majority showing an anxiety state as found in the series in the United States.[2] A classification of the types of psychoneurosis is as follows: conversion symptoms or hysteria, 18; anxiety state, 18; hypochondriasis, 7; unclassified or mixed, 4; and constitutional psychopathic state, 4.

In the differential diagnosis, organic types of joint disease, including arthralgia and orthopedic conditions, were chiefly considered. Rheumatoid arthritis furnished some difficulty even after a careful examination and an adequate period of observation. Several patients were seen, however, who had been admitted with a diagnosis of neurosis and whose symptoms were found to be entirely explain-

able on the basis of rheumatoid arthritis with marked constitutional manifestations. In a larger number — 10 of 132 patients at the Sixth General Hospital with rheumatoid arthritis — two diagnoses were properly made, with the coincident neurosis usually of the anxiety type. In some cases the arthritis apparently became worse after the development of the neurosis, but in at least one soldier who had carried on in spite of a well marked arthritis, the anxiety was obviously secondary to the disability and to a feeling of helplessness in the presence of danger. Since the diagnosis of hypertrophic arthritis was made largely by means of x-ray examination in the Theater, patients chiefly required mention whose symptoms were out of all proportion to the organic findings. Such patients, if there was positive evidence of neurosis, were usually given two diagnoses, one of which included "psychoneurosis, manifested by elaboration of a minor organic illness." Others, who showed hypertrophic changes only incidentally or with presenting symptoms entirely unrelated, were also given two diagnoses, with the lack of importance of the arthritis clearly indicated. Points found useful in the distinction between arthralgia and psychogenic rheumatism have been outlined above. As a final check, even in apparently clear-cut cases of neurosis, it was well to have an orthopedic examination to eliminate structural changes of the skeletal system as a partial or complete cause of the patient's disability.

Treatment and Course

The treatment of psychogenic rheumatism was largely unsuccessful once the patient reached a general hospital, whether the symptoms were long present, were precipitated by combat or the hardships of military life or became localized and fixed during previous hospitalization. About all that was accomplished was to reassure the patient (and the medical officer) that no serious form of arthritis was present and to determine the disposition according to the patient's degree of disability. An even more pessimistic conclusion was reached by Boland and Corr,[2] who found that their efforts to return such patients to full or limited duty were largely unsuccessful. Their group, however, was made up of men undergoing training in the United States, and in many cases the only solution was the removal of the underlying mental conflict, which could be accomplished by separation from military service. The authors stress the need for prompter recognition and proper psychotherapy instead of physical therapy, a point that was equally applicable, as mentioned above, to the Theater. For example, it was found possible at the Six Hundred and First Clearing Company to send most of the patients with hysterical manifestations localized to the skeletal system at least to limited service by early application of treatment before the symptoms became intensified and fixed. The methods consisted in the clearing up of symptoms with the aid of barbiturate narcosis and suggestion. The patients were usually told in addition that their combat days were over but that prolonged and unpleasant hospitalization was in store for them unless they became well enough for limited service.

Disposition

In the series of 51 patients with psychogenic rheumatism from the Twelfth General Hospital, only 4 (8 per cent) were returned to duty, 30 (60 per cent) were sent to limited service and 17 (32 per cent) were evacuated. Although it was agreed that disposition by general hospitals should be individualized according to the degree of neurosis and an estimate of the patient's ability to perform useful service, the impression was gained that the majority were discharged to limited service. A few, especially those with mild or exaggerated symptoms, were sent to full duty, such a disposition being more probable in forward medical echelons. The remainder, usually with a severe underlying neurosis and a deep fixation of symptoms, were evacuated.

Summary

Hypertrophic arthritis was encountered in the Mediterranean Theater as frequently as rheumatoid arthritis, a finding associated with the presence of older officers and soldiers and a high incidence of the secondary type. Symptoms usually existed prior to induction, the hardships and physical strain of military service being precipitating factors in hospitalization. Previous joint trauma or fractures were responsible for the development of the secondary type about three times as often as structural abnormalities. The diagnosis of hypertrophic arthritis was established by x-ray examination, with care necessarily taken to ascertain the relation of the x-ray findings to the patient's disability. The proportion of patients sent to limited service was greater than that in patients with rheumatoid arthritis.

Reasons are advanced for the use of the term "arthralgia" in patients with symptoms referred to the joints, muscles and other supporting structures, without evidence of a true arthritis or a causative neurosis. The high incidence of cases of arthralgia is stressed. Symptomatic treatment was effective only in the acute and localized forms, in which the diagnosis of myositis or myalgia was often made. Over 50 per cent of these patients were returned to full duty, and another 25 per cent to limited service.

At least a sixth of the patients with articular complaints were found to have a neurotic basis for their symptoms — so-called "psychogenic rheuma-

tism." Articular localization of psychosomatic manifestations was less frequent than backache, headache and gastrointestinal or cardiovascular symptoms. Precipitation or aggravation of the neurosis by combat was frequent, and many patients gave a history of previous joint disease or injury. About half the patients had additional generalized or localized functional symptoms. Treatment was ineffective except in patients seen soon after the onset of symptoms in a forward area.

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CLINICAL NOTE

MONGOLISM FOLLOWING INTERCURRENT INFECTIOUS DISEASE IN PREGNANCY*

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IT IS generally agreed that mongolism is not hereditary in the sense that hemophilia and color blindness are hereditary.[1, 2] Abundant evidence[1] shows that the risk of mongolism is greater in mothers of advanced age than in those who are younger. There is also a higher frequency of mongolism in homozygote as contrasted with heterozygote twins.[3] By some this is interpreted to mean that the ovum or sperm is defective. Early reports tending to implicate diseases like syphilis, tuberculosis, malaria and alcoholism[2] are inadequate to explain the large majority of cases. Indeed, no one etiology has been shown for the condition.

No cases have been reported that correlate minor maternal illnesses with the subsequent development of the condition. The recent discovery that maternal rubella may be followed by such widely disseminated and such severe fetal consequences as congenital cataract, anomalies of the heart, microcephaly and mental retardation[4, 5] has brought about a sudden change of perspective to both pediatricians and obstetricians. This change has to do with their point of view toward maternal infections that were once considered too trivial in their effect on the mother to have any bearing on fetal health. With this changed opinion in mind, we report 7 cases of intercurrent infectious disease observed during pregnancies that terminated in the delivery of a mongoloid baby. These are listed in Table 1. The localization of the particular disease in 6 of the 7 cases at about the end of the second month of pregnancy is provocative. It is more so in view of the long recognized association of mongolism with such congenital deformities as cleft palate, syndactily and imperforate anus, which date from about the same period of fetal life.[6] We believe that further epidemiologic studies are indicated to show whether acute maternal infections correlate with the subsequent development of mongolism.

Even if acute intercurrent infectious disease proves to be one factor in the etiology of mongolism, it probably accounts for only a small fraction of all cases. The problem of demonstrating such a factor is not dissimilar to that posed by the relation of tonsillectomy to subsequent poliomyelitis, tonsillectomy being shown to be a factor in a small fraction of all cases (about 1 per cent). The relation was demonstrated not by the ratio of cases of poliomyelitis to tonsillectomies but by a significant pyramiding of the developing cases ten to twenty days postoperatively.[7, 8]

In determining the relation of intercurrent infectious disease to mongolism, the week of pregnancy in which the particular disease occurred is of statistical importance. Since there are forty weeks to a normal pregnancy, there is only one chance in forty for a given illness to occur in any particular week — if the phenomenon is a chance one. When a sufficient number of cases are assembled the figures can be submitted to statistical analysis.

TABLE 1. *Data on Mothers of Mongoloid Babies Who Had Had Infectious Diseases during Pregnancy.*

AGE OF MOTHER	INTERCURRENT DISEASE	STAGE OF PREGNANCY
yr.		mo.
25	Influenza	2
34	Rubella	2
39*	Mumps	2
43(?)†	Mastoiditis	2
39	Pleurisy (in bed 3 wk.)	3
35‡	"Grippe"	6
23§	Acute purulent otitis media and sinusitis (with vomiting)	2

*The information on this case was furnished by Dr. Richard C. Tefft, Jr.
†Mother had pneumonia at birth of child and died.
‡On a diet during first month of pregnancy patient lost 15 pounds.
§Sinusitis occurred just prior to otitis. Mother was admitted to hospital because of vomiting necessitating intravenous administration of fluids.

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We hope that this brief communication will prompt physicians to provide us with further data.

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MEDICAL PROGRESS

PATHOLOGY*

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FOUR years have elapsed since I have had the privilege of recording progress in pathology for this series. During that period staffs of civilian laboratories were cut to the bone, and the demands of routine diagnostic work and year-round teaching under the accelerated program of medical education left little opportunity or energy for investigative work. To a considerable extent this was balanced by the necessity of pathological investigation of problems of military importance, a need largely cared for in this country by the farsighted policy of the United States Army in developing and progressively expanding the Army Institute of Pathology with its large corps of full-time workers and its part-time consultants. The unparalleled collection of anatomic material that has flowed into it from every corner of the globe has already permitted many studies of importance. A portion has already been published, publication of much more is imminent and study of the remaining material will occupy as large a corps of investigators as can be maintained for years to come.

Any attempt to survey the entire field of pathology for a four-year period would require such cursory treatment as to be completely valueless. A few features only have been selected because they seem to be of more than casual interest and importance.

Rheumatic Fever and Periarteritis Nodosa

One of the most interesting conceptions in pathology in recent years is the hypothesis advanced by Rich[1] that the lesions of periarteritis nodosa and rheumatic fever are the result of anaphylactic hypersensitivity. The possibility that rheumatic fever is a hypersensitive reaction had been entertained for many years by a variety of investigators, and the early literature was reviewed by Swift[2] and Gross,[3] in 1929, and again by Coburn[4] in 1931. Gross repeated many of the reported experiments with negative results. In succeeding years Klinge,[5] Vaubel[6] and Junghans[7] each claimed to have produced lesions of the rheumatic type by sensitization of animals with horse serum, but their published illustrations were not convincing. Bruun,[8] in 1940, repeated their experiments with only equivocal results and concluded that he had not reproduced a true analogue of the Aschoff nodule.

In 1942 Rich[9] reported autopsies on 5 patients who had had serum sickness shortly before death, in all of whom vascular lesions characteristic of periarteritis nodosa were demonstrable. None of the patients had any symptoms suggestive of periarteritis prior to the acute terminal illness, and all the vascular lesions were fresh. Similar vascular lesions were also demonstrated in 2 patients who had had reactions to sulfonamide therapy.[10] He concluded that vascular lesions of the periarteritic type can be a manifestation of the anaphylactic type of hypersensitivity.

In the following year Rich and Gregory[11] reported the experimental production of similar lesions. Rabbits were sensitized with one or more large doses (10 cc.) of horse serum. On the fifth to the eighth day the animals developed fever and had a slight blushing of the skin, possibly equivalent to the urticaria of human serum sickness. All animals showed on skin test the immediate, anaphylactic type of hypersensitivity, some developing Arthus reactions with necrosis. They were sacrificed at periods ranging from seventeen to twenty-six days, and the majority showed arteritic lesions characteristic of various stages of periarteritis nodosa.

In an extension of this experiment the same authors[1] found in the hearts of 11 of 36 similarly sensitized rabbits lesions that reproduced closely the five supposedly pathognomonic features of acute rheumatic carditis. These are focal alterations of

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collagen, Aschoff nodules, focal and diffuse inflammatory lesions, focal alterations in cardiac muscle and verrucose valvular vegetations. In the experimental animals connective-tissue alteration was prominent in the endocardium near the valvular attachments, and nodules developed about the foci of damaged collagen composed of large cells with abundant usually basophilic cytoplasm, of which some were multinuclear, and others resembled Anitschkow myocytes. In a few nodules palisading of cells about the altered collagen was observed. These nodular foci were usually formed near the attachments of the valves. Perivascular inflammatory infiltrates were frequent, but nodule formation in the adventitia of arteries — a characteristic location in rheumatic fever — was not observed. Nonspecific inflammatory lesions in the endocardium and pericardium were frequent. In 4 animals small foci of necrosis of cardiac muscle fibers were found. No actual vegetations with thrombus formation were noted, although the nodules frequently projected above the surface of the valve to give it a slightly warty appearance. The parallelism traced between the experimental lesions and the histologic findings in acute rheumatic fever is impressive, if not complete, and the excellent illustrations guarantee the objectivity of the verbal descriptions. Evidence for the anaphylactic character of still another rheumatic lesion was furnished by the same authors by a demonstration of the essential similarity of the pulmonary injury following sulfonamide sensitization and that of rheumatic pneumonitis.[12]

The greater success of Rich and Gregory, in comparison with their predecessors, in reproducing histologic features closely simulating those of rheumatic fever in human beings may well have been due to their clear distinction between the anaphylactic and tuberculin types of hypersensitivity and their selection of more appropriate sensitizing regimes. There can be little hesitation over accepting their hypothesis regarding periarteritis nodosa, in which clinical evidence of hypersensitivity, such as eosinophilia or asthmatic paroxysms, is frequently present. Even here, caution is suggested by the reports of periarteritis developing in rats and dogs made hypertensive by clamping of the renal artery[13] or by wrapping of the kidney in silk.[14] Despite the close parallelism between the symptomatology of rheumatic fever and serum sickness the usual stigmas of allergy are lacking in the former disease, and direct proof of anaphylactic sensitization in rheumatic fever in the human being has yet to be advanced. Studies such as those of Coburn[4] with streptococcal antigens should be repeated with attention to immediate rather than delayed types of skin reaction.

Acute Disseminated Lupus Erythematosus

Acute disseminated lupus offers so many general features of similarity to and so many details of difference from rheumatic fever that comparison of the two diseases fascinates clinicians and pathologists alike. A new angle of comparison has been advanced by Klemperer, Pollack and Baehr[15] in a continuation of the pathological studies on lupus that have occupied the laboratory of the Mt. Sinai Hospital for many years. On the basis of a review of the 35 cases that have come to autopsy in that institution, with particular study of the last 20, they have reached the conclusion that the thesis formerly advanced — that lupus is fundamentally a diffuse vascular disease[16] — is no longer tenable. They now present evidence that the basic pathologic lesion is a characteristic type of focal connective-tissue degeneration. All elements take part in the reaction. The mucoid ground substance, ordinarily hardly visible, becomes evident as a swollen homogeneous interfibrillar mass. The collagenous fibers assume a straight, rigid, irregularly thickened appearance and become deeply eosinophilic, assuming the character often designated as fibrinoid. The fibroblasts show degeneration, proliferation and even necrosis. Inflammatory reaction, although variable, is nonspecific in type and relatively inconspicuous, and in the authors' words, "the alterative phase of the process is far out of proportion to the exudative." This is in marked contrast to rheumatic fever, a disease in which collagenous degeneration has long been recognized as one of the most important pathologic features, but in which active inflammatory reaction of a specific character about the foci of altered collagen is constant. The involvement of serous cavities is considered secondary to the connective-tissue degeneration of the limiting membranes, rather than to primary mesothelial involvement as Gross[17] suggested. Again, the first change noted in the endocardium and valves was in the connective tissue rather than in the endothelium. The so-called "wire-loop" changes of the glomerular tufts in the kidney are evidently a primary change of the collagen of the capillary basement membranes, and even the focal glomerulitis of other cases can be so interpreted. Vascular lesions, although seen most often in the kidneys, are observed rather frequently in other organs. The mildest recognizable change is a deposit of fibrinoid material within the intima, and later between the muscle fibers of the media and in the adventitia. It is usually accompanied by fibroblastic proliferation. Endothelial damage is definitely considered secondary, in contradiction to previous work from this same laboratory that considered it primary.[16] In severe lesions secondary necrosis of the entire vascular wall may occur, closely simulating the vascular lesions of malignant hypertension or even periarteritis nodosa.

Connective-tissue changes of the type described are much less conspicuous in the skin, although they do occur. It is doubtful if many dermatopathologists would admit that they were primary. In the spleen, fibrinoid degeneration was unusual, but another form of connective-tissue change was found

in 19 of 20 cases. This consists of periarterial fibrosis limited to central and penicilliary arteries in the form of concentric rings of dense collagen fibers. This lesion is sufficiently specific to have considerable diagnostic value. Unaccounted for by the theory of primary collagenous injury is the extensive involvement of lymphoid tissue frequently observed.

Rheumatoid Arthritis

Joint lesions so dominate the clinical picture of rheumatoid arthritis that comparatively scant attention has been given to the possibility of involvement of other organs and tissues. The possibility of cardiac involvement has been raised by Baggenstoss and Rosenberg.[18, 19] Cardiac lesions indistinguishable from ordinary rheumatic carditis were found in several cases of rheumatoid arthritis, and in 2 the histologic picture was similar to that of rheumatoid nodules. Steiner et al.[20–22] have recently shown that peripheral nerves and muscles are frequently affected. The lesions in both nerves and muscle consist of small aggregates of mononuclear cells, lymphocytes and plasma cells predominating but with occasional macrophages. There is usually no evidence of any core of necrotic collagen as in the rheumatoid nodules, and no palisades of epithelioid cells develop. The foci are found in the perineurium and perimysium of nerves and muscles respectively. Since there is no evidence of axonal or myelin-sheath degeneration, it seems improbable that nerve function is impaired. In the muscles, atrophy was frequently observed, but the evidence for attributing this to the inflammatory foci is not convincing.

In discussion of Steiner's[22] paper Clauson reports the results of biopsies of the deltoid muscle from 70 cases of rheumatoid arthritis. In more than half he was able to find lesions similar to those described by Steiner. He also found indistinguishable lesions in a few cases of rheumatic fever.

Atypical Pneumonia

One of the great advantages of a central institute of pathology, such as that fostered by the Army in Washington, is in the study of the pathology of diseases with a low mortality. Few hospital laboratories, for instance, have had the opportunity of studying more than one or two examples of atypical pneumonia. The Army Institute of Pathology has autopsy material from 90 cases in which that diagnosis had been made on clinical grounds. Many were inadequately studied, and others were complicated by secondary bacterial infection, but Golden[23] was able to select 21 entirely acceptable cases for analysis. In this group of cases focal bronchiolitis was a constant finding. The lumens contained frank pus, mucoid secretions and desquamated epithelial cells, sometimes in an advanced stage of disintegration. Ulceration of the mucosa was not rare. Bacteria were absent in most cases, and when present were neither numerous nor uniform in type. The affected bronchioles were dilated, and their walls were infiltrated with mononuclear cells. This inflammatory infiltrate tended to spread radially into the adjacent tissues, such as the alveolar walls and the interlobular septums. The alveoli were in part aerated, focally atelectatic and occasionally filled with protein-rich edema from which hyaline membranes precipitated on the alveolar walls. They were always relatively free from polymorphonuclear infiltration. Fibrinous pleuritis was unusual, in contrast to its frequency in bacterial pneumonias. In cases of longer duration foci of squamous-cell metaplasia frequently appeared in the bronchi, and the alveoli sometimes became lined with columnar epithelium.

The type of pneumonitis described may fairly be classed as an interstitial pneumonitis. As McCordock and Muckenfuss[24] pointed out, viruses that involve the lung tend to produce this type of pneumonitis. Interstitial pneumonitis cannot, however, be considered reliable evidence of virus infection, since it occurs in conditions as different as scrub typhus, Q fever and even histoplasmosis.[25]

Scrub Typhus

Until the American invasion of the South Pacific, scrub typhus was an unknown disease to most American physicians, and few would even have been able to identify it with the fascinatingly alliterative tsutsugamushi, which had lingered as a mere name among the vaguer recollections of medical-school days. From the material of the Army Institute of Pathology, Allen and Spitz[25] have been able to describe thoroughly the pathology of the disease and to compare the lesions with those of the related rickettsioses: epidemic typhus, Rocky Mountain spotted fever and Q fever.

A noticeable primary lesion, known as the eschar, usually develops at the point of the mite bite. This is often frankly suppurative and probably represents a combined reaction to the inoculated rickettsias and to the irritating secretions of the mite. In contrast to epidemic typhus and still more so to spotted fever, there is relatively little evidence of vascular damage in scrub typhus. Cerebral involvement is qualitatively similar to that in epidemic typhus, but the lesions are less numerous and the individual nodules are somewhat smaller and less conspicuous. Interstitial myocarditis, in contrast, is more constant and severe in scrub typhus than in either the epidemic disease or in spotted fever. In view of the intensity of the interstitial reaction, remarkably little evidence of muscle-cell involvement is observed. Endocardial involvement was found in a third of the cases. Interstitial pneumonitis of marked degree is not rare in scrub typhus, whereas it is seldom evident in epidemic typhus or spotted fever. In this respect the disease shows an apparent relation to Q fever, in which pneumonitis

is often the predominating feature. One other point of differentiation appeared in the lymph nodes, which frequently show necrosis in scrub typhus but rarely do so in the other rickettsial diseases.

Injury Due to Cold

The recent war offered abundant opportunities for studying injury due to exposure to low temperatures. Several closely related syndromes have been described under such titles as "trench foot,"[26] "immersion foot"[27] and "high-altitude frostbite"[28] in American, British and German journals. The similarities of clinical and pathological findings are great, and the differences inconstant, suggesting that the underlying tissue injury is similar regardless of the type of exposure and whether or not actual freezing of the tissue occurs. The most complete pathological study to date is that of Friedman[26] from the Army Institute of Pathology based on 14 cases of trench foot. The findings closely parallel those of Siegmund[29] in cases of frostbite on the German-Russian front and of Blackwood[27] in cases of immersion foot.

The initial reaction following cold injury of all types is a prolonged period of vasoconstriction. This is followed by a stage of intense hyperemia. In the majority of severe cases this is accompanied by edema and frequently by exudation and hemorrhage into the tissues. In severe cases necrosis develops, leading to wet or dry gangrene. Pathological investigation has been hampered because surgery is delayed as long as possible, and most amputation material has shown only late stages of the process. Friedman was fortunate in having at his disposal several extremities from men dying of other causes shortly after developing trench foot. He found in this early material constant changes in the skin, subcutaneous fat, blood vessels and nerves. In the skin intercellular and intracellular edema was found in the malpighian layer in the earliest stages; later, vesiculation or frank necrosis and mummification occurred. The skin appendages were often involved, particularly the coil glands. Degeneration of collagen and elastic fibers, which was frequent in the later stages, was inconstant in the earlier ones. Involvement of the deeper portion of the subcutaneous fat tissue was constant and surprisingly extensive in view of earlier reports. In acute stages leukocytic infiltration was prominent, and edema and even fibrinous exudation into the interlobular fibrous septums were observed. Later, atrophy of fat lobules, accumulation of foam cells and replacement fibrosis became frequent. Occasionally, oil cysts formed. It seems probable that the loss of this protective layer had much to do with the long persistent functional disability in some cases that escaped gangrene. Vascular abnormalities were constant. In the early stages extreme dilatation and engorgement of small vessels were evident, frequently in association with perivascular hemorrhages. The next stage of involvement was the appearance of thrombotic masses of agglutinated red cells, relatively free from platelet debris and fibrin in comparison with ordinary thrombi. Angiitis, although usually present, was not particularly severe. In later stages (thirty to forty days), organization of thrombi and endarterial intimal proliferation were usual. Muscle changes were inconspicuous in the early cases, but extensive atrophy was noted in late lesions. Nerves in the early cases showed swelling, edema and focal degeneration of axis cylinders and myelin. In later stages demyelinization and perineurial fibrosis were observed. White and Warren[30] suggested that the latter was responsible for the persistent pain noted in certain cases. It was not possible, on the basis of the pathological evidence, to decide whether vascular or neural changes were primary.

Epidemic Hepatitis and Homologous Serum Jaundice

The pathology of epidemic hepatitis was comparatively unknown until the outbreak of World War II. Jaundice due to massive necrosis of the liver had been recognized as early as 1842 by Rokitansky,[31] who first described and named "acute yellow atrophy." It was, however, regarded as a uniformly fatal disease. Catarrhal jaundice was considered a benign and totally unrelated malady. Although the Scandinavians Flindt,[32] Linstedt,[33] Ehrström[34] and Wallgren[35] and the Englishman Cockayne[36] successively pointed out at intervals from 1890 to 1930 the parallelism of incidence between catarrhal jaundice and atrophy of the liver, general medical opinion was essentially uninfluenced by their observations. The world-wide pandemic of hepatitis in the soldiers of all belligerent countries in the recent war has provided unlimited material for study. From the spring of 1942 to April, 1945, material from 296 fatal cases was submitted to the Army Institute of Pathology. The first 118 of these were reported by Lucké[37] in 1943. The succeeding 178 cases were subsequently reviewed by Lucké and Mallory.[38]

Most fatal cases of hepatitis can be readily classified into one of two types, subacute and fulminant, which show corresponding variations in clinical and pathological findings. The subacute type was exemplified by the series of cases developing in the American Army in 1942, which was reported by Lucké. None of the patients died in less than ten days, a few survived for one hundred days or more, and the survival period in 60 per cent ranged from twenty to fifty days. In these cases the liver showed extensive but irregularly distributed necrosis and numerous, often large, yellow or green foci of regeneration. This is the condition that for years was classified as "idiopathic acute yellow atrophy." In the second series of cases, reviewed by Lucké and Mallory, the majority (53 per cent) of patients died in less than ten days, many in as short a period

as three or four days, and a few even before the onset of jaundice. In such cases the symptomatology is often predominantly cerebral. The hepatic necrosis is diffuse and massive and no evidence of regeneration is seen, but the process has occurred so fast that the liver has had little time to shrink and is often only slightly reduced in size. Frequently, the gross appearance is that of an exaggerated "nutmeg liver." Cases of this type have also been reported by Wood[39] in material derived from personnel of the United States Navy. Despite the difference in the two types, enough points of similarity and enough transitional forms are observed to make it certain that they are not two diseases but represent a wide range of reaction to the same fundamental disorder.

The significant pathology of the disease is centered in the liver. In both types two features are constantly found — extensive degeneration and necrosis of liver cells and inflammatory reaction, most evident in an infiltration of the periportal connective tissue, with histiocytes and smaller numbers of other wandering cells. The necrosis is widespread and uniform in the fulminant cases, and regeneration is minimal or absent. Of particular interest are the patients dying in two to four days, either before or shortly after the onset of jaundice. Total destruction of the liver parenchyma has already occurred, and it is difficult to escape the conclusion that severe hepatic involvement must have antedated the first clinical symptoms.

It is of interest to compare the clinicoanatomic classification of hepatitis described above with current epidemiologic classifications. Four types of the disease are recognized from the latter point of view: the sporadic and epidemic forms of the "naturally" acquired disease, homologous serum jaundice and arsphenamine hepatitis. No clinical differences have been established and studies of nonfatal cases by the biopsy technic[40–42] have likewise failed to provide grounds for distinction. Luckė and Mallory found that a majority of their fulminant cases occurred in wounded men who had received plasma or whole-blood transfusions from fifty to ninety days before the onset of symptoms of hepatitis. This range is within the accepted incubation period of homologous serum jaundice. On the other hand, nearly a third of the fulminant cases appeared in men who had never received infusions of human plasma. These findings were balanced, however, by Luckė's cases in 1942, all of which were of the subacute variety despite the fact that the majority were cases of homologous serum jaundice secondary to yellow-fever vaccination. It is evident that the spontaneous and the homologous serum cases may each run either a subacute or a fulminant course and that the method of infection is not the explanation of variations in clinicoanatomic type. If different viruses are involved,[43, 44] they produce identical end results.

Since this report was submitted numerous other important studies from the Army Institute of Pathology have been published.[45]

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

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CASE 33121

PRESENTATION OF CASE

First admission. A sixty-four-year-old English wool dealer was admitted to the hospital because of severe nausea and vomiting of one day's duration. He had had no pain, hematemesis or any change in bowel habits.

Physical examination was entirely negative except for a blood pressure of 170 systolic, 95 diastolic. Two urine examinations showed a +++ test for albumin with many white cells and bacteria in the sediment. Examination of the blood showed a hemoglobin of 14.2 gm. and a white-cell count of 11,200, with 84 per cent neutrophils. A gastrointestinal series revealed a diverticulum projecting medially from the second portion of the duodenum. Recuperation was spontaneous, and the patient was discharged four days after admission.

Final admission (eight months later). During the night before admission the patient became restless and nauseated but did not vomit. At 7:30 a.m. a severe, sharp pain, "like a rifle shot," developed under the "breast bone" and in the throat. This was associated with shortness of breath, and about four hours later the pain radiated down the arms and into the back between the scapulas.

Physical examination revealed a pale man who was slightly obese but not short of breath. The pulse was 112 and of good quality, and the blood pressure was 128 systolic, 90 diastolic. The neck veins were distended and pulsating while the patient was lying down. The heart did not appear enlarged. The aortic second sound was equal in intensity to the pulmonic second sound, and there were no murmurs. The cardiac sounds were somewhat decreased in intensity. The chest was clear, and the abdomen normal. There was slight cyanosis of the fingernails, but no peripheral edema. An electrocardiogram three and a half hours after the onset of the attack was within normal limits. At 1:30 p.m. on the day of admission the patient had a convulsion, the pulse became weak and irregular and the blood pressure could not be obtained. Oxygen and 0.5 cc. of adrenalin were administered, but the pulse finally became imperceptible and the patient expired.

DIFFERENTIAL DIAGNOSIS

DR. CONGER WILLIAMS: From the data presented, I cannot reach any conclusion about the illness that caused the first hospital admission, and I doubt whether this episode had any relation to the final acute illness. Possibly, the urinary findings can be explained by urinary infection, although a widespread disease affecting both the kidneys and the heart, such as periarteritis nodosa, should not be overlooked. The one value of the examination at the first admission is to establish the fact that the blood pressure was slightly elevated. It may be of some importance in determining the cause of the final episode.

At this point it is well to consider what conditions caused the acute pain in the chest. First of all, in a man of this age, the possibility of coronary heart disease with acute infarction must always be considered. In favor of that diagnosis is the fact that the pain was located beneath the sternum and radiated up into the throat, later going into the arms. This may have happened when the pain increased in intensity. The drop in the systolic blood pressure to 128 from the former level of 170 is also consistent with infarction. Against the diagnosis is the quality of the pain, which was described as sharp. The characteristic pain of myocardial infarction is dull or squeezing, often described as a sensation of pressure. Some patients in describing pain, however, use the adjective "sharp" to mean severe rather than knifelike. It is possible that some confusion arose merely from the choice of terms in description. Another point against the diagnosis of infarction is the apparent suddenness of onset of severe pain, although the restlessness during the preceding night may have marked the real beginning of the trouble. Also, whereas radiation of pain into the back occurs with myocardial infarction in a fair percentage of cases, I think that radiation of pain from the sternum into the neck, down both arms and into the back — during the course of one attack — is rather unusual. Repeated attacks may occur in which the radiation of pain

varies, but I believe that this was an unusually wide referral of pain for a case of infarction.

Certainly, in any acute emergency giving rise to chest pain, the possibility of dissecting aortic aneurysm should be considered. In the first place we know that this patient had hypertension, which can almost be called a necessary prelude to dissection. Secondly, the onset and intensity of the pain in the chest are both more in favor of dissecting aneurysm than of myocardial infarction. If it is assumed that the first pain, described as substernal and radiating into the neck, was produced by involvement of the mouths of the coronary arteries in the progress of dissection, it might be said that the pain in the back, instead of being referred pain, represented extension of the dissection along the course of the aorta down into the descending portion. Another point that should be mentioned and is perhaps slightly against the diagnosis although not ruling it out is the fall of the blood pressure. Usually, in acute dissection the blood pressure is surprisingly well sustained. I shall say more about that later.

Apparently, in the history a statement was made about shortness of breath. That should be clarified. The patient may have meant that he had sufficient substernal discomfort to produce a sensation of shortness of breath, which is quite different from dyspnea. I assume that he did not have dyspnea because it was not evident on physical examination.

The finding of distended and pulsating neck veins is not necessarily significant with the patient in the horizontal position. The fact that this man may have had increased venous pressure should be considered, however, and its implications should not be overlooked. If he actually had an increase in peripheral venous pressure, what conditions were responsible? I should like to mention three as being the likeliest. The first is congestive heart failure, for which evidence is practically nonexistent in this case. Although shortness of breath was mentioned, it was not observed on physical examination. The lungs were clear, and we have nothing except this one finding to suggest failure of the myocardium. Another condition that produces an acute, sudden increase in peripheral venous pressure is cardiac tamponade. This, of course, is a frequent complication of dissection of the aorta that progresses down through the root of the aorta, with rupture into the pericardial sac. Also, the break may occur above the base of the aorta but below the attachment of the pericardium to the aorta. Therefore, that is a possibility. If the leak is small, death does not necessarily ensue immediately — the patient may survive for several hours. If there is an increase in venous pressure as a result of acute cardiac tamponade, pulsus paradoxicus is usually evident on examination. It is possible to miss a paradoxical pulse, however, if the measurement is made simply by palpation of the radial artery. It is sometimes necessary to use auscultation to detect pulsus paradoxicus. Acute pericardial tamponade would also explain the fall in blood pressure. The third possibility to explain the increase in venous pressure is pulmonary embolism. I find nothing to support that diagnosis, and if this man had pulmonary embolism, the widespread pain must be explained on the basis of a secondary myocardial infarction. Pulmonary embolism complicated by myocardial infarction is not too rare, but I discard that possibility in this case.

The normal electrocardiogram is slightly against the diagnosis of myocardial infarction but by no means rules it out. It is possible to have a large infarction with no change in the electrocardiographic findings for hours or even days.

There are several possibilities to explain the sudden exitus. Dissection could have involved the cerebral vessels, producing convulsions. Or this may have been caused by cardiac arrest, either from auriculoventricular block — the so-called "Adams-Stokes phenomenon" — or from ventricular fibrillation. Another possibility is a sudden massive hemorrhage that, by producing acute cerebral anoxia, may also have caused the convulsions. A frequent termination of aortic dissection is rupture into the pleural cavity, with massive hemorrhage. Besides aortic aneurysm and myocardial infarction, I considered the other conditions that one usually thinks of in the differential diagnosis of acute chest pain. Those included acute pancreatitis, which I discarded because of the absence of abdominal findings and also because of the absence of vomiting on the final admission. I do not believe that any of the acute pulmonary episodes, such as pulmonary collapse, pneumothorax and mediastinal emphysema, explain the history as given or the radiation of pain into the arms, neck and back.

In conclusion my diagnosis is dissecting aortic aneurysm, mostly on the basis of the history. I realize that several of the positive findings one likes to see, such as the rapid development of an aortic diastolic murmur and absent pulsations in one or more of the limbs, were not present.

DR. BENJAMIN CASTLEMAN: Have you anything to add, Dr. White?

DR. PAUL D. WHITE: The fact that the neck veins were pulsating as well as distended helps to rule out any large mass, such as hemorrhage, compressing the mediastinal vessels. In such a case I should expect that there would be distention but not pulsation in the vessels.

CLINICAL DIAGNOSIS

Dissecting aneurysm of aorta?

DR. WILLIAMS'S DIAGNOSIS

Dissecting aneurysm of aorta.

Anatomical Diagnoses

Dissecting aneurysm of aorta, with rupture into pericardium.
Cardiac tamponade.
Chronic pyelonephritis: left kidney.

Pathological Discussion

Dr. Castleman: Autopsy showed everything that Dr. Williams predicted. This was a dissecting aortic aneurysm. The intimal tear had occurred about 1.5 cm. above the aortic valve and had dissected proximally down around the coronary mouths. Although at the time of autopsy little blood was found in the dissecting channel, there was separation down to and around the coronary mouths, which may explain the anginal attacks. The aneurysm had extended distally along the arch and the descending aorta into the left iliac artery. It surrounded and extended into one of the renal arteries. The external tear was not easy to make out, but we know that it occurred within the pericardium because the pericardial cavity was filled with blood. The fact that we could not find the actual tear indicates that there may have been a slow leak over a period of two or three hours. That might account, as Dr. Williams suggested, for the distention of the neck veins. Another interesting finding — one that we have seen in a number of cases with pericardial hemorrhage — was extravasation of blood into the adventitia of the pulmonary arteries. This blood extended along and formed a hemorrhagic extravasation in the subpleural space, especially in the interlobar septums. This may also have added to the distention of the neck veins.

The only other finding of note was a chronic left pyelonephritis, which probably accounted for the urinary findings found at the final admission.

Dr. Howard B. Sprague: There is one point of interest in this case that I wish to bring out. I did not see the patient myself, but asked Dr. William C. Bridges to see him on an emergency call. I discovered later that this man had attended an annual banquet of the wool trade on the previous night. His friends told me that he appeared perfectly well and that they could not understand how such a healthy person could be dead the next day. He was active in business, making frequent trips abroad, and was free from symptoms. The incident tends to spread the belief among laymen that heart disease strikes without warning. As a matter of fact, most patients dying of coronary disease have warnings of an anginal character for some time prior to the fatal attack. Lack of such symptoms is further evidence, therefore, in favor of dissecting aneurysm as against coronary occlusion, although the rule does not hold in every case. Whether or not the heavy banquet was a factor in precipitating the aortic rupture, one cannot say.

Dr. White: It is of some importance, particularly in treatment, to differentiate as soon as possible myocardial infarction and dissecting aortic aneurysm. From the standpoint of prognosis the chances are ten to one of recovery from myocardial infarction and ten to one against recovery from a dissecting aneurysm. That is approximate, of course.

CASE 33122

Presentation of Case

A fifty-year-old woman was admitted to the hospital because of palpitation, dyspnea and orthopnea.

She had been treated by a physician for high blood pressure and "sick headaches" for many years. Five years before entry, because of profuse menstrual periods and intermenstrual bleeding, a hysterectomy had been performed for fibroids of the uterus. Adrenalin given preoperatively caused palpitation. After operation the physician told the patient that she must have had rheumatic fever in childhood because the aortic valve was "all eaten away" and the heart was "greatly swollen." An attack of phlebitis in the left leg subsequently forced the patient to remain in bed for a month. At that time she began to have orthopnea and exertional dyspnea and palpitation. There was no ankle edema, cyanosis or cough. Digitalis was administered, and the dyspnea cleared; the patient was able to return to her household duties after a month or two. She continued to notice exertional dyspnea and palpitation, however, and was forced to lead a rather sedentary life. Her condition remained unchanged until four months before admission, when the lower teeth were extracted, and a week or two later there was a sudden brief attack of nausea, a dry hacking cough, soreness in the ribs and fever. The pounding of the heart became marked, and a severe ache developed in the right knee and calf, extending to the toes, which became swollen. The pain in the knee cleared a few days later, but there was persistence of fever and malaise. The patient was hospitalized and given penicillin, with considerable relief, but the nausea persisted when she returned home. She remained on a liquid diet because of inability to swallow solid food. She had frequent green loose stools that started while she was in the hospital and persisted despite the fact that for years previously she had taken daily laxatives because of constipation. She returned to the hospital, where x-ray studies of the stomach, bowel and gall bladder were reported as negative, and penicillin was again given for a week. She continued to have palpitation, exertional dyspnea, night sweats, orthopnea and dysphagia. A week before admission exquisite tenderness developed in the left calf and ankle and the left toes. The patient stated that, according to her physician, in the few weeks prior to admission the blood pressure had dropped to normal.

The past history revealed no definite rheumatic fever, chorea or scarlet fever. An operation for a

ruptured appendix had been performed about fifteen years previously. The patient had had two pregnancies; the first child died in early infancy, and the second did not survive a cesarean section. A sister had died of what had seemed to be carcinoma of the stomach.

On physical examination the apical impulse was 9 cm. from the midsternal line in the fifth interspace. There were a high-pitched, Grade III apical systolic murmur, a loud blowing systolic murmur at the base, loudest at the aortic area and transmitted to both sides of the neck, and a systolic thrill in the aortic and pulmonic areas. One observer heard a diastolic murmur in the pulmonic area. There were scars of the right rectus muscle beneath which a vague doughy mass that felt like adhesions was noted. Marked tenderness in the right upper quadrant just beneath the costal margin prevented palpation of the liver, but to percussion the liver did not appear enlarged. Light palpation in the right upper quadrant elicited rebound tenderness in the left upper quadrant. Scattered small red lesions, a few of which resembled petechiae, were noted in the skin. A small petechia with a white center was noted beneath the conjunctiva of the right lower lid. The left leg was warmer than the right. Both calves were tender when squeezed, the left more so than the right, and the left ankle was swollen and hot, without pitting edema. The ankle was quite tender. The toes of the left foot were markedly red and exquisitely tender, with erythematous patches, measuring 1 to 2 mm. in diameter, over the left ankle and lateral aspect of the foot. Homans's sign was negative. There was no limitation of motion, and adequate pulsations were felt in the dorsalis pedis arteries bilaterally. There was questionable early clubbing of the fingers and toes.

The temperature was 99.6°F., the pulse 100, and the respirations 22. The blood pressure was 110 systolic, 64 diastolic.

Examination of the blood revealed a red-cell count of 5,150,000, with a hemoglobin of 13 gm., and a white-cell count of 10,200, with a normal differential count. The urine was normal. Plain films of the chest and abdomen were normal; the liver margin appeared normal. The total protein was 7.3 gm., the nonprotein nitrogen 30 mg., the uric acid 3.5 mg. and the fasting blood sugar 107 mg. per 100 cc. An electrocardiogram was normal on the fifth hospital day.

While the patient was in the hospital the blotches and the tenderness subsided somewhat, and a subsequent observation revealed absent pulsations of the right dorsalis pedis artery but equal pulsations were felt in the popliteal and femoral arteries on both sides. Blood cultures taken on the fourth hospital day grew colonies of alpha-hemolytic streptococcus. A week later the clubbing of the fingers became more pronounced, but no new petechiae developed. On the twelfth hospital day examination of the heart revealed a Grade III aortic systolic murmur, with a thrill, and a Grade II aortic diastolic blow over the left sternal border, with no definite separate mitral murmurs. The hemoglobin at that time was 8.5 gm. per 100 cc. Penicillin was given in a continuous drip starting on the sixteenth hospital day. On the eighteenth hospital day, however, the temperature rose to 103.5°F., and the patient was somewhat sleepy but euphoric when aroused. There were no localizing neurologic signs, but the following morning the patient complained of numbness of both hands, and there was some diminished tactile and position sense, especially on the left. She seemed somewhat confused throughout the day and spilled a glass of water on two occasions, presumably from the left hand. A test of the alpha-hemolytic streptococcus isolated by blood culture revealed that it grew in 0.25 units of penicillin per cubic centimeter and was inhibited by 0.5 units per cubic centimeter. Consequently, the penicillin dosage was increased to 1,000,000 units a day by intramuscular drip. Two days later the hands had cleared considerably, and a week later the patient seemed to be recovering. No new symptoms had developed.

The patient continued to run a temperature, occasionally as high as 101.5°F. during the next few weeks, and on the forty-first hospital day she developed intermittent abdominal cramps, which were largely periumbilical, and by evening she vomited 400 cc. of watery material. Examination revealed no acute abdominal tenderness or tympany, and peristaltic rushes could be heard concomitant with the pain. The temperature was 103.5°F., and the white-cell count 13,000. A plain film of the abdomen on the next day revealed distended loops of small intestine and a suggestion of a soft-tissue mass in the pelvis above the bladder shadow, more on the left than on the right, at which site the distention of the small bowel stopped. Fecal material was noted in the colon. Re-examination after the use of a Harris tube revealed the small intestine to be markedly deflated. Because of the continued pain and a rise in the white-cell count to 17,700, an exploratory laparotomy was done and adhesions were lysed. She appeared to recover from the operation, but spiking fevers continued on occasion. On the sixth postoperative day she had a mild headache, with numbness of one finger, and fever. On the following day the patient woke at 1:00 a.m. yelling incoherently. She was disoriented and recognized no one. She vomited recently ingested material once and put her hand to the left side of her head in a manner suggesting pain in this area. The neck was slightly stiff. The pupils were equal; the right pupil reacted sluggishly to light. The motor power in the extremities seemed equal, and she reacted to painful stimuli. No abnormal reflexes were encountered. She died about six hours later. The last positive blood culture had been obtained on the fifteenth

hospital day, and twelve subsequent blood cultures had been negative.

Differential Diagnosis

Dr. James H. Currens: The symptoms of palpitation, dyspnea and orthopnea are indicative of heart disease, probably due to limited myocardial reserve.

After the operation a physician told the patient that she must have had rheumatic fever in childhood because the aortic valve was "all eaten away" and the heart was "greatly swollen." This was the first evidence of valvular heart disease and was discovered when the patient was forty-six years of age.

It is of interest that she had phlebitis of the left leg as a complication of the hysterectomy. Phlebitis, particularly on the left, is not a rare complication of a pelvic operation, particularly hysterectomy. Whether she had pulmonary emboli at that time to account for the increase in exertional dyspnea and palpitation is difficult to determine. Certainly, the history is not specific on that point.

The past history indicated a decreased cardiac reserve, probably on the basis of a decreased reserve of the left ventricular myocardium over a period of five years. The appearance of fever two weeks after a dental extraction is important. The fact that she developed fever and did poorly after the extraction of several teeth is a strong indication of the hazard of dental extraction in the presence of valvular heart disease. It is our practice to give penicillin as a prophylactic measure to people with valvular and congenital heart disease before and for at least twelve hours after dental extractions. I think that cough may be a somewhat neglected symptom in heart disease, but I fail to associate the cough in this patient with the subsequent events.

It would be of interest to know whether the patient had pain with the diarrhea. Presumably, she had none. The night sweats are probably significant. I interpret them as evidence of continued infection. Tenderness in the left calf in the presence of a history of phlebitis of the left leg strongly suggests the recurrence of a thrombophlebitis in that leg.

One wonders if the previous hypertension was merely systolic hypertension, possibly associated with some nervousness and palpitation of the heart. The diastolic blood pressure of 64 is certainly normal, but in view of the findings it may have indicated a mild degree of aortic insufficiency.

"The apical impulse was 9 cm. from the midsternal line in the fifth interspace." Unfortunately, we do not know the position of the midclavicular line. Since the impulse was in the fifth interspace, however, there was probably relatively little cardiac enlargement. I think that the auscultatory findings were quite characteristic of aortic stenosis, as was the transmission of the systolic murmur to the apex. The presence of a thrill in the pulmonary area is somewhat unusual, but if the thrill is strong enough, it could be palpated well over the sternum and presumably on both sides of the sternum. One observer heard a diastolic murmur in the pulmonic area. That is probably a significant finding in view of the subsequent clear-cut aortic diastolic murmur.

The vague doughy mass that felt like adhesions was a remarkable observation, but in view of the subsequent development of intestinal obstruction, it presumably was correct.

In any cardiac patient with pain or tenderness in the right upper quadrant one must think of liver congestion. There is not much evidence in the physical findings, however, to suggest either systemic or pulmonary congestion. Tenderness in the left upper quadrant, although the spleen was not palpable, may have been due to splenic infarction. The other possibility, I suppose, is a mycotic aneurysm, but it is rather difficult to see how a mycotic aneurysm of the lower aorta or mesenteric artery could account for such pain.

The observation of scattered small red lesions, a few of which resembled petechiae, is important if the diagnosis of subacute bacterial endocarditis is considered, and there is evidence strongly in favor of such a diagnosis. I should interpret the pain in the left calf as having been due to phlebitis of the leg. Homans's sign was negative. That sign, however, is usually an early indication of phlebitis of the leg, so that it might easily be absent later on. The finding of a petechia beneath the conjunctiva of the lower lid is an important positive sign when one is considering bacterial endocarditis. The same thing should be said of the early clubbing of the fingers and toes.

The laboratory data were normal throughout except for the borderline hemoglobin.

It is unusual for an x-ray film of the heart to be normal in a person with the symptoms that this patient had. I think that we had better see the x-ray films.

Dr. Richard Schatzki: In this film, which was taken ten weeks before death, the heart is not enormously enlarged, but I think that the left ventricle looks rounder than usual, as if there was some left ventricular hypertrophy.

Dr. Currens: Certainly, the heart was not greatly enlarged, and it makes me question to a large degree many of the symptoms that the patient had had in the past five years. Left ventricular failure does not occur with a normal-sized heart. She may well have had some cardiac neurosis complicating the organic heart disease.

The disappearance of the pulsation of the right dorsalis pedis artery, with good pulsations above, suggests a small embolus in the right lower leg.

The anemia may explain the more apparent aortic diastolic murmur that developed under observation. Another explanation might be ulceration of the aortic-valve leaflets to allow greater insufficiency of the valve.

On the eighteenth hospital day the temperature rose to 103.5°F., and the patient was somewhat sleepy but euphoric when aroused. A high temperature is an unusual finding in the presence of penicillin therapy. These findings are evidence, I should think, of embolism to the brain, resulting in "numbness of both hands and diminished tactile and position sense, especially on the left."

"A test of the alpha-hemolytic streptococcus isolated by blood culture revealed that it grew in 0.25 units of penicillin per cubic centimeter and was inhibited by 0.5 units per cubic centimeter." This indicates a moderately resistant organism and may or may not have been affected by the previous penicillin that the patient had received on two occasions.

I do not know how the patient died, since the record does not state what happened during the last six hours. She may have died in coma.

The primary diagnosis is certainly that of aortic valvular heart disease, with both stenosis and regurgitation. Although there was no history of rheumatic fever, rheumatic heart disease is the best bet so far as the etiology of the aortic stenosis is concerned. She was a little young for aortic stenosis presumably resulting from arteriosclerosis. I believe that this type of aortic stenosis is more frequent in men than in women. Another possibility is congenital heart disease with a bicuspid aortic valve. Perhaps the patient was too old for that, although patients with congenital bicuspid valvular disease live fairly long and not infrequently develop stenosis or regurgitation, or both.

The evidence for subacute bacterial endocarditis seems clear cut: fever, petechiae, anemia and bacteremia. There is good evidence for embolic phenomena to the brain and the right leg. The absence of splenomegaly certainly does not stand in the way of such a diagnosis. The complications of subacute bacterial endocarditis are of importance to consider. In patients who have been treated with penicillin in the last few years it has been possible to cure the bacterial infection in 65 to 70 per cent of cases if a sufficient amount was given. In 30 or 35 per cent of fatal cases, the patients die primarily of complications of one type or another, possibly of heart failure. The infection is rarely completely resistant if heroic doses of penicillin are used. In this case only 1,000,000 units of penicillin a day was given. If the infection is impossible to control, it is essential to increase the penicillin as much as possible. Doses as high as 30,000,000 or 40,000,000 units a day have been used. In this hospital I think that the largest dose used has been 10,000,000 units a day. Once penicillin is started it should be continued for at least three or four weeks. The two courses of penicillin for a week each before entry to this hospital were obviously ineffective in eradicating the infection, although they suppressed it temporarily.

The evidence for peripheral emboli is good. In bacterial endocarditis of the aortic valve, emboli occasionally enter a coronary artery.

I believe that this woman had aortic valvular disease with superimposed bacterial endocarditis and with embolic phenomena. It has been my impression that patients with bacterial endocarditis of the aortic valve are likelier to have emboli than those with infection on the mitral valve. The possibility of a mycotic aneurysm of the aorta, a cerebral artery, a mesenteric artery or an artery of the extremity remains good. A disturbing factor in this patient is that she continued to have fever and evidence of peripheral emboli. Mycotic aneurysms usually, of course, manifest themselves by rupture, resulting in a subarachnoid hemorrhage when the aneurysm is located in a cerebral artery.

Another possibility is that of active rheumatic fever. Perhaps 10 or 20 per cent of the cases with subacute bacterial endocarditis and rheumatic heart disease have evidence of active rheumatic fever during the course of the disease or at autopsy. It is conceivable that some of the pain in the left ankle and the continued fever were on the basis of active rheumatic fever. The joint pains, however, were not impressive, and one might expect evidence of more heart failure with active rheumatic fever. The electrocardiogram was normal, which is against active rheumatic fever, serious myocardial disease or hypertrophy. The evidence is quite good that the infection persisted either on the aortic valve or in an unrecognized mycotic aneurysm.

The fact that the organism was moderately resistant also suggests that penicillin did not cure the infection. Against such a diagnosis is, of course, the fact that the blood cultures were negative. It is conceivable that the infection had been suppressed temporarily by the penicillin but not eradicated. This observation was not infrequent in the use of the sulfonamides before the penicillin era, at which time the blood cultures often remained negative for days or weeks only to become positive again. Another possibility is a mixed bacterial infection of the aortic valve, which has been described by Orgain and Poston*; conceivably penicillin might have eradicated only one of the organisms. I should interpret the final outcome in this patient as having been due to a persistence of infection either of the aortic valve or in a mycotic aneurysm.

She may or may not have had pulmonary emboli. The history is certainly not suggestive.

DR. EDWARD F. BLAND: A low-grade fever during penicillin therapy sometimes disappears on cessation of the therapy and hence need not necessarily imply continued infection. Furthermore, even embolism may occur after the infection has been arrested.

*Orgain, E. S., and Poston, M. A. Mixed infections in bacterial endocarditis. *Am. Heart J.* 23:823-836, 1942.

DR. CURRENS: Have you ever seen a temperature of over 100°F. with penicillin therapy for bacterial endocarditis?

DR. BLAND: I believe so.

CLINICAL DIAGNOSES

Subacute bacterial endocarditis.
Rheumatic heart disease.
Cerebral embolus.

DR. CURRENS'S DIAGNOSES

Rheumatic heart disease, with aortic stenosis and regurgitation.
Bacterial endocarditis, with either persistent infection or mycotic aneurysm.

ANATOMICAL DIAGNOSES

Subacute bacterial endocarditis: aortic valve.
Cerebral mycotic aneurysm, with subarachnoid hemorrhage.

PATHOLOGICAL DISCUSSION

DR. TRACY B. MALLORY: Post-mortem examination showed a normal-sized heart, but there was evidence of old rheumatic heart disease — slight on the mitral valve and quite definite on the aortic valve, with enough deformity to have produced stenosis and probably a little regurgitation. There were vegetations on the aortic valve that were larger and more pliable than one sees in purely rheumatic heart disease but not so active as those observed in a subacute bacterial endocarditis. The immediate cause of death was an extensive subarachnoid hemorrhage. It is rather interesting that we found few manifestations of embolism. One can almost count on infarcts of the spleen and kidneys in cases of bacterial endocarditis. Both these organs were entirely free from any gross evidence of embolism. We could not tell whether histologically the vegetations on the aortic valve showed evidence of healing, since we decided to sacrifice the histologic studies for bacterial studies. The entire material of the valve was ground up and extensively cultured, but we were unable to grow out any organisms from the valve substance, so that it appears to have been bacteriologically sterile.

DR. SCHATZKI: How thick was the left ventricular wall?

DR. MALLORY: Of normal thickness. The heart weighed only 300 gm. — a little on the small side.

DR. JAMES H. TOWNSEND: What was the cause of the subarachnoid hemorrhage?

DR. MALLORY: There was a mycotic aneurysm.

DR. BLAND: Were there any Aschoff bodies?

DR. MALLORY: No.

The New England
Journal of Medicine

Formerly
The Boston Medical and Surgical Journal

Established 1828

Owned by the Massachusetts Medical Society and Published under the Jurisdiction of the Committee on Publications

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Subscription Terms: $6.00 per year in advance, postage paid, for the United States (medical students, $3.50 per year); Canada, $7.00 per year (Boston funds); $8.50 per year for all foreign countries belonging to the Postal Union.

Material should be received not later than noon on Thursday, two weeks before date of publication.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to the *New England Journal of Medicine*, 8 Fenway, Boston 15, Massachusetts.

THE PROBLEM FAMILY IN BRITAIN

A recent editorial[1] expresses British concern for a lamentable social evil, the so-called "problem family" — a term used to describe a segment of the population sunk in poverty, ignorance and degradation, incapable of responding to improving influences and providing a reservoir of delinquency, vice and disease. Perhaps mindful of the record of Government participation in medical care and public health elsewhere in the British Commonwealth,[2] the editorial, although recognizing the need for some measure of corrective social legislation, points out that the apparatus for ameliorative action already exists and that a voluntary organization — the Society of Friends — has demonstrated an effective approach to the situation: treating the family as a unit, reconciling estranged members, rehabilitating the home and restoring self-respect. It endorses Brockington's[3] program for reclaiming the problem family from hopeless destitution and degeneracy; this solution combines the authority of the magistrate and the personal, humanitarian approach of the social worker — the former places the family on probation and introduces the social worker, with whom the family co-operates in the hope of successfully completing the period of probation and thereby of retaining the children.

Brockington, observing that a great number of homeless children had been separated from their families because of parental cruelty, neglect and immorality, destitution, illegitimacy and lack of parental control, recommended that the problem family be reconstituted and placed under the supervision of health authorities before reaching the stage of enforced separation, and that authority be granted for registration of the problem family, rehabilitation of the home and appointment of special staffs for teaching and supervision. He further suggested that current legislation, with its tendency to break up the family, be amended to empower magistrates to remove the family as a unit to care and protection after the failure of probation, and emphasized the fact that delinquent children should be separated from their families only after child guidance and efforts to improve home conditions have failed.

The interest of the British medical profession in the problem family is to be commended. The constructive recommendations for prevention and amelioration without extensive social legislation merit careful attention in the United States, where similar families exist in disquieting plenty and where governmental paternalism is ever expanding. In medical sociology, as in other fields, organized medicine can influence the course and extent of Government intervention.

References

1. Editorial. Problem families. *Lancet* 1:928, 1946.
2. Clemons, M. G. W. Changing face of medical practice in Tasmania. *M. J. Australia* 1:609-611, 1946.
3. Brockington, C. F. Homelessness in children: causes and prevention: analysis of unparented children in three English counties. *Lancet* 1: 933-936, 1946.

PREVENTABLE CANCER

ALTHOUGH it is true that the cause of cancer is unknown in most cases, it is equally true that the etiology in certain cases is obvious and that such cancers are therefore preventable. Into the latter class fall the occupational cancers arising in persons as the result of regular and prolonged contact with physical or chemical carcinogenic agents.

Such occupational neoplasms as the soot cancer of chimney sweeps and the cancer of the lung occurring in the Schneeberg miners have long been recognized. That malignant tumors of the bladder occur in workers in the aniline dye industry in the United States was not realized until 1932, and the fact that leukemoid conditions leading to death sometimes follow exposure to benzol even in small amounts was not appreciated until even more recently. Cancer of the lung has been noted among workers in chromate plants and nickel refineries and among asbestos workers. Furthermore, it is said that cancer of the lung kills 75 to 80 per cent of unprotected cobalt workers, and carcinogenic properties have been found in such varied substances as arsenic, tar, crude mineral oil, shale oil, certain aromatic amines and, of course, roentgen rays and rays from radioactive substances.

It has been recommended that the medical profession, as well as employers and employees, be better educated regarding such carcinogenic agents and that special care be taken to guard the worker against them.* Medical supervision of those exposed to carcinogenic agents seems to be a wise step, and it is possible that some form of governmental inspection of industrial plants in which carcinogenic agents are used should be instituted. For example, an intelligent college graduate who was clearly suffering from early myeloid metaplasia was recently seen by a physician who is particularly cognizant of these industrial hazards. The man categorically denied that benzol was used in the plant in which he worked, yet inspection by the proper state authorities a few days later showed that the plant reeked with benzol and that no precautions were being taken. Such conditions should not exist.

In connection with all occupational cancers it should be remembered that many years of exposure to a given carcinogenic agent may elapse before the malignant neoplasm makes itself manifest, and this very fact is likely to make both the employee and the employer lax in the matter of precautions taken. One should also remember that there are many individual variations in the reactions of those exposed. Thus, in a family of four, all of whom were equally exposed to benzol over an equal period, one member died of myeloid metaplasia, one of a leukemoid disorder, and one of pancytopenia; the fourth member remained perfectly well, so far as could be ascertained.

Since an increasing number of carcinogenic agents are being found, both in the home and in industry, it behooves all of us to guard against their untoward reactions.

*Hueper, W. C. Industrial management and occupational cancer. *J. A. M. A.* 131:738-741, 1946.

MASSACHUSETTS MEDICAL SOCIETY
POSTWAR LOAN FUND

The Postwar Loan Fund has been set up, and all discharged medical officers who were members of the Massachusetts Medical Society in good standing at the time of their entry into the service may apply for loans from this fund. For further information apply to:

George L. Schadt, *Chairman*
Postwar Loan Fund
8 Fenway
Boston 15, Massachusetts

MASSACHUSETTS MEDICAL SOCIETY

COMMITTEE ON ETHICS AND DISCIPLINE

Owing to a recent episode reported in the newspapers and involving one of the fellows of the Society, who suffered because of strict adherence to his interpretation of the "Code of Ethics" and the "Oath of Hippocrates," the following observation is offered.

Chapter II, Section 1, of the "Principles of Medical Ethics of the American Medical Association," reads as follows:

Patience and delicacy should characterize all the acts of a physician. The confidences concerning individual or domestic life entrusted by a patient to a physician and the defects of disposition or flaws of character observed in patients during medical attendance should be held as a trust and should never be revealed *except when imperatively required by the laws of the state.* . . . (Italics mine.)

The physician's oath requires strict observance of the above quotation, but physicians are citizens of the state in which they reside, bound to observe its laws, bound to do all in their power to promote the public good and bound to give such aid to the courts that justice may prevail.

RALPH R. STRATTON, *Chairman*

POSTGRADUATE LECTURE COURSE

The program for the remainder of the Postgraduate Lecture Course held at Sanders Theater in Memorial Hall, Harvard University, Cambridge, under the sponsorship of the Committee on Postgraduate Medical Education, Massachusetts Medical Society, in co-operation with the Massachusetts Department of Public Health is as follows:

Monday, March 31. OBSTETRICS. *Chairmen*: Roy J. Heffernan and Frederick C. Irving.

5:30–5:50. **Diet in Pregnancy.** Bertha L. Burke: assistant professor of maternal and child nutrition, Harvard School of Public Health.

5:50–6:20. **The Rh Factor.** Louis K. Diamond: assistant professor of pediatrics, Harvard Medical School; visiting physician, Children's Hospital.

6:20–6:40. **New Developments in Prenatal Care.** Charles L. Sullivan: assistant obstetrician, St. Elizabeth's Hospital and Massachusetts General Hospital.

6:40–7:00. **Induction of Labor.** Roy J. Heffernan: visiting obstetrician and gynecologist, Carney Hospital; consulting obstetrician and gynecologist, Faulkner Hospital and St. Margaret's Hospital.

7:00–7:30. **Buffet Supper.**

7:30–7:50. **The Use of X-Ray in Obstetrics.** Daniel J. McSweeney: assistant professor of obstetrics, Boston University School of Medicine and Tufts College Medical School; visiting obstetrician, Boston City Hospital and St. Margaret's Hospital.

7:50–8:10. **The Fetus during Labor.** Charles L. Sullivan.

8:10–8:30. **Extrauterine Pregnancy.** Daniel J. McSweeney.

8:30–8:50. **Indications for Forceps Delivery.** Roy J. Heffernan.

Wednesday, April 2. GASTROENTEROLOGY. *Chairmen*: Franz J. Ingelfinger and Chester M. Jones.

3:00–3:25. **Procedures in Diagnosis and Management of Peptic Ulcer.** Franz J. Ingelfinger: associate professor of medicine, Boston University School of Medicine; chief, Gastrointestinal Outpatient Department, Massachusetts Memorial Hospitals; member of staff, Evans Memorial, Massachusetts Memorial Hospitals.

3:25–3:50. **Medical Management of Peptic Ulcer.** Sara M. Jordan: director of gastroenterology, Lahey Clinic; physician, New England Baptist and New England Deaconess hospitals.

3:50–4:15. **Surgical Management of Peptic Ulcer.** Francis D. Moore: associate in surgery, Harvard Medical School; assistant surgeon, Massachusetts General Hospital.

4:15–4:30. **Intermission.**

4:30–5:05. **Diagnostic and Medical Measures Pertinent to the Problem of Painless Jaundice.** Chester M. Jones: clinical professor of medicine, Harvard Medical School; physician, Massachusetts General Hospital.

5:05–5:40. **Surgical Measures in Relation to Painless Jaundice.** Reginald H. Smithwick: professor of surgery, Boston University School of Medicine; surgeon-in-chief, Massachusetts Memorial Hospitals; member of board of consultants, Massachusetts General Hospital.

5:40–6:00. **Discussion.**

Monday, April 7. PART I. OFFICE PROCEDURES IN ANORECTAL DISEASE. *Chairmen*: E. Parker Hayden and Neil W. Swinton.

5:30–5:45. **Differential Diagnosis of Rectal Bleeding and Methods of Rectal and Colon Examinations.** E. Parker Hayden: chief, Proctologic Clinic, and assistant visiting surgeon, Massachusetts General Hospital.

5:45–6:00. **Hemorrhoids.** Roy E. Mabrey: assistant surgeon, Massachusetts General Hospital.

6:00–6:10. **Anal Fissure.** Roy E. Mabrey.

6:10–6:25. **Anorectal Abscess and Fistula.** E. Parker Hayden.

6:25–6:40. **Premalignant Lesions of the Rectum and Colon.** Neil W. Swinton: surgeon, Lahey Clinic, New England Deaconess and New England Baptist hospitals.

6:40–7:00. **Anal Pruritus.** Neil W. Swinton.

7:00–7:30. **Buffet Supper.**

PART II. RADIATION THERAPY. *Chairmen*: Joseph H. Marks and Samuel A. Robins.

7:30–7:45. **Contact Therapy of Superficial Lesions.** Bernard E. LeVine: radiation therapeutist, Beth Israel Hospital.

7:45–8:00. **Hodgkin's Disease.** Hugh F. Hare: director, Department of Radiology, Lahey Clinic.

8:00–8:15. **Fibroids and Menorrhagia.** Samuel A. Robins: professor of radiology, Tufts College Medical School; chief radiologist, Beth Israel Hospital and Evangeline Booth Hospital.

8:15–8:30. **Carcinoma of the Cervix.** Laurence L. Robbins: roentgenologist, Massachusetts General Hospital.

8:30–8:45. **Carcinoma of the Ovary.** Joseph H. Marks: instructor in radiology, Harvard Medical School; radiologist, New England Deaconess and Palmer Memorial hospitals.

8:45–9:00. **Discussion.**

Wednesday, April 9. DISEASES OF BLOOD AND LYMPH NODES. *Chairmen*: William Dameshek and Thomas H. Ham.

3:00–3:30. **Diagnosis of Anemia.** Thomas H. Ham: assistant professor of medicine, Harvard Medical School; associate physician, Thorndike Memorial, and junior visiting physician, Boston City Hospital.

3:30–4:00. **Treatment of the Anemias.** Maurice B. Strauss: chief of medical service, Cushing General Hospital, Framingham.

4:00–4:35. **Diagnosis and Treatment of Disorders of the Spleen.** William Dameshek: professor of clinical medicine, Tufts College Medical School; hematologist, Pratt Diagnostic Hospital; chief, Blood Clinic, Boston Dispensary.

4:35–4:45. **Intermission.**

4:45–5:15. **Treatment of Leukemia.** Henry Jackson, Jr.: associate physician, Thorndike Memorial, Boston City Hospital; assistant professor of medicine, Harvard Medical School.

5:15–5:45. **Transfusion Therapy.** Charles P. Emerson: assistant professor of medicine, Boston University School of Medicine; member of staff, Massachusetts and Evans Memorial, Massachusetts Memorial Hospitals.

5:45–6:00. Discussion.

Monday, April 14. THE PREVENTION OF VENOUS THROMBOSIS AND PULMONARY EMBOLUS. *Chairmen*: Arthur W. Allen, James A. Evans and John Homans.

5:30–6:00. **Clinical Features of Pulmonary Embolism.** Lewis Dexter: assistant in medicine, Harvard Medical School; senior associate in medicine, Peter Bent Brigham Hospital.

6:00–6:30. **Experiences with Heparin in Pitkin's Menstruum Combined with Dicumarol in the Treatment of Venous Thrombosis.** James A. Evans: physician, Lahey Clinic; physician, New England Baptist and New England Deaconess hospitals.

6:30–7:00. **Anticoagulant Drugs versus Vein Ligation in the Treatment of Thrombophlebitis at the Beth Israel Hospital.** Arnold Starr: junior visiting surgeon, Beth Israel Hospital.

7:00–7:30. **Buffet Supper.**

7:30–8:00. **Three-year Study of Rest, Exercise and Early Ambulation Relative to Venous Thrombosis: Analysis of 2000 consecutive cases.** James C. McCann: surgeon, St. Vincent's Hospital, Worcester.

8:00–8:30. **Prophylactic Femoral-Vein Division at the Massachusetts General Hospital.** Robert R. Linton: visiting surgeon and chief, Peripheral Vascular Clinic, Massachusetts General Hospital; associate in surgery, Harvard Medical School.

8:30–9:00. **The Prophylactic Use of Dicumarol in Gynecology.** George Van S. Smith: professor of gynecology, Harvard Medical School; chief, Free Hospital for Women.

Wednesday, April 16. PART I. KIDNEY DISEASE. *Chairmen*: Reginald Fitz and W. Richard Ohler.

3:00–3:30. **Diagnosis of Renal Disease.** Laurence B. Ellis: associate in medicine, Harvard Medical School; assistant visiting physician, Boston City Hospital.

3:30–4:30. **Treatment of Renal Disease.** Reginald Fitz: lecturer on the history of medicine, Harvard Medical School; consulting physician, Peter Bent Brigham Hospital; and W. Richard Ohler, assistant professor of medicine, Harvard Medical School; chief, Second Medical Service, Boston City Hospital.

4:30–4:35. **Intermission.**

PART II. HYPERTENSION. *Chairmen*: Robert S. Palmer and Reginald H. Smithwick.

4:35–5:00. **The Heart in Hypertension.** Paul D. White: clinical professor of medicine, Harvard Medical School; physician, Massachusetts General Hospital.

5:00–5:30. **The Value of Various Therapeutic Measures in the Management of Hypertension and Hypertensive Cardiovascular Disease.** Robert S. Palmer: associate physician, Massachusetts General Hospital; consultant, Massachusetts Eye and Ear Infirmary.

5:30–6:00. **Surgical Measures in Hypertensive Patients.** Reginald H. Smithwick: professor of surgery, Boston University School of Medicine; surgeon-in-chief, Massachusetts Memorial Hospitals.

Monday, April 21. CARDIOVASCULAR DISEASES. *Chairmen*: Samuel A. Levine and Howard B. Sprague.

5:30–6:00. **Pericarditis.** Howard B. Sprague: instructor in medicine, Harvard Medical School; associate physician, Massachusetts General Hospital; chief of medicine, House of the Good Samaritan.

6:00–6:30. **The Diagnosis of Coronary-Artery Disease.** James M. Faulkner: professor of medicine, Tufts College Medical School.

6:30–7:00. **The Treatment of Cardiac Emergencies.** Paul D. White: clinical professor of medicine, Harvard Medical School; physician, Massachusetts General Hospital.

7:00–7:30. **Buffet Supper.**

7:30–8:00. **The Clinical Features of Patent Ductus Arteriosus.** Samuel A. Levine: assistant professor of medicine, Harvard Medical School; physician, Peter Bent Brigham Hospital.

8:00–8:30. **The Heart in Acute Infections.** Charles A. Janeway: professor of pediatrics, Harvard Medical School.

8:30–9:00. **The Treatment of Subacute Bacterial Endocarditis.** Chester S. Keefer: professor of medicine, Boston University School of Medicine; director, Evans Memorial, Massachusetts Memorial Hospitals.

Wednesday, April 23. PEDIATRICS. *Chairmen*: Stewart H. Clifford and Richard C. Tefft, Jr.

3:00–3:45. **Influenza Meningitis: Diagnosis and treatment.** John A. V. Davies: assistant professor of pediatrics, Harvard Medical School.

3:45–4:30. **Factors Influencing the Growth and Development of the Fetus.** Clement A. Smith: assistant professor of pediatrics, Harvard Medical School.

4:30–4:40. **Intermission.**

4:40–5:15. **Influence of Disease on the Growth and Development of the Child.** Stuart S. Stevenson: associate in child health, Harvard Medical School and Harvard School of Public Health.

5:15–6:00. **Influence of Endocrine Dysfunction on Acceleration or Retardation of Growth and Development in the Child and Adolescent.** Nathan B. Talbot: assistant professor of pediatrics, Harvard Medical School.

MEDICOLEGAL ABSTRACT

Relation of Patient and Physician: Liability for malpractice. The defendant attended the plaintiff during childbirth, and the latter had a long period of labor followed by a difficult delivery. Before the plaintiff was placed on the delivery table Scott's solution was applied to the legs and buttocks. While she was on the delivery table she lay on her back, the buttocks resting on a rubber mat covered by a sterile towel. She was on the delivery table for more than one hour, during which time the defendant first performed an episiotomy and then accomplished the delivery with the use of a forceps.

After the operation the plaintiff complained of a burning sensation over the buttocks. The defendant examined her and found second-degree burns over an area of about 5.0 by 7.5 cm. on each buttock. The defendant examined the plaintiff and, on discovering the burns, exclaimed: "My God, what a mess; my God, what happened here. . . . It is a darn shame to have this happen." He also said, "She had a very hard delivery, and it was a burning shame to get that on top of it, and it was because of negligence when they were upstairs." The burns

eventually developed keloids. Later the defendant told the plaintiff's husband that it was unfortunate, that a thing like that should not have happened and that the staff was going to take steps to correct it, and that apparently it had been caused by the solution's having been allowed to stay on the mat, thus exposing the skin, for too long a period. There was testimony by an expert called by the defendant that the burns were due to friction and pressure, that the use of Scott's solution was accepted obstetric practice and that the plaintiff's injuries were unavoidable.

The trial judge directed a verdict for the defendant on the ground that there was no evidence from which the jury could find that the defendant had been negligent. On appeal the Supreme Judicial Court said in part:

> The jury could have found, in accordance with the defendant's admission, that the plaintiff's injuries were due to negligence; that there was no negligence on the part of the plaintiff or of any one other than the defendant; that, by the process of elimination, this negligence was that of the defendant. . . . It is likewise no answer to say that the admissions were merely statements of regret, sympathy and benevolence evoked by human suffering.

The court ruled that the case should have gone to the jury, and the trial judge having directed a verdict for the defendant, a new trial was granted. (*Woronka* v. *S*——, Mass. Adv. Sh. [1946] 1207.)

CORRESPONDENCE

NEED FOR DIPHTHERIA IMMUNIZATION

To the Editor: The number of cases of diphtheria in the City of Boston has continued to increase steadily. The Boston Health Department is making every effort through the press, radio and personal contact to notify all who are charged with the care of children that the number of cases is mounting and is alarmingly high and that immediate action must be taken to control the disease.

In the six years preceding 1946, there were 179 cases, with 10 deaths. In 1946, the corresponding numbers were 157 and 10. So far this year, 66 cases of diphtheria have developed in Boston. This is by far the greatest number of cases that has occurred in any two months during the last fifteen years. It indicates a trend in diphtheria that calls for an aggressive immunization program throughout the city.

This month, a city-wide campaign for diphtheria immunization is being conducted in Boston. Practicing physicians are urged to emphasize the need for diphtheria immunization and booster doses. The Boston Health Department is advocating that all children be taken to their own physicians or, where this is not practical, to the various clinics provided by the Health Department and by school authorities.

Studies indicate that children who have had original inoculations against diphtheria should be furnished booster doses of toxoid at intervals. The Boston Health Department in its clinics uses 0.5 cc. of the toxoid supplied by the Massachusetts Department of Public Health, injected subcutaneously. This toxoid is available free of charge at all City of Boston culture stations.

JOHN H. CAULEY, M.D., M.P.H., *Health Commissioner*
Health Department
Haymarket Square
Boston 14

BOOK REVIEWS

The Extremities. By Daniel P. Quiring, Ph.D.; Beatrice A. Boyle; Erna L. Boroush, M.A.; and Bernardine Lufkin, A.B. 8°, cloth, 117 pp., with 105 illustrations. Philadelphia: Lea and Febiger, 1945. $2.75.

The simple line drawings of the origin, insertion, nerve and blood supply of the muscles of the extremities presented in this volume should be of great service to students and surgeons alike. It seems to the reviewer regrettable that the references are to Gray and Cunningham only, with none to the excellent textbook by Morris. This book is a product of the joint efforts of earnest students at the Cleveland Clinic Foundation. The extensive experience of the authors should serve well the needs of those interested in the functional principle of muscle action.

Physical Chemistry of Cells and Tissues. By Rudolf Höber, M.D.; J. B. Bateman, Ph.D.; David R. Goddard, Ph.D.; and Wallace O. Fenn, Ph.D. 8°, cloth, 676 pp. Philadelphia: Blakiston Company, 1945. $9.00.

This is an extremely interesting and instructive book whose contents convincingly prove that physiology is deeply anchored in physical chemistry. The volume is arranged in sections as follows: selective principles of physical chemistry; large molecules: their physicochemical properties and their architectural and functional significance in living matter; introductory remarks concerning the architecture of protoplasm; the surface of the protoplast, its properties and its architecture; influence of some extracellular factors on cellular activity; the respiration of cells and tissues; contractility; and passive penetration and activity transfer in animal and plant tissues.

The volume has an excellent index of authors and subjects. Each chapter of each section is documented by excellent references. This should be a most useful book for physicists, chemists and physiologists.

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

The Vitamins in Medicine. By Franklin Bicknell, D.M., M.R.C.P.; and Frederick Prescott, M.Sc., Ph.D., A.R.I.C., M.R.C.S., clinical research director, Wellcome Foundation, London. Second edition, revised and enlarged. 8°, cloth, 916 pp., with 208 illustrations. New York: Grune and Stratton, 1946. $12.00.

This new edition of an English book has been revised by the rewriting and expansion of the chapters on the vitamin B complex and riboflavin. The other chapters have been brought up to date with the advances in knowledge of the vitamins. A new chapter has been added on the essential unsaturated fat-soluble acids (vitamin F) and minor fat-soluble vitamins. The references to the literature have been increased to four thousand five hundred, and the number of illustrations almost doubled. The printing is excellent, with a good type on good paper. A comprehensive subject index concludes the volume. This treatise should prove valuable as a reference source.

The Modern Attack on Tuberculosis. By Henry D. Chadwick, M.D.; and Alton S. Pope, M.D., deputy commissioner of public health, and director of the Division of Tuberculosis, Commonwealth of Massachusetts. Revised edition. 8°, cloth, 134 pp. New York: Commonwealth Fund, 1946. $1.00.

This monograph was first published in 1942 and reprinted twice previous to this edition, which has been revised in the light of recent developments in administrative practice and in the technics of case finding and control. New material has been added, and some sections expanded, including photofluorography, the role of mass x-ray examination in industry and other population groups, with emphasis on the

essential role of an organized follow-up system, the financial aspects of hospitalization, rehabilitation, chemotherapy and the federal case-finding program. The volume is recommended for all persons interested in the tuberculosis problem and in public health.

A B C of Medical Treatment. By E. Noble Chamberlain, M.D., M.Sc., F.R.C.P., lecturer in medicine, University of Liverpool, physician to outpatients, Royal Infirmary, Liverpool, and visiting physician, Smithtown Road Hospital. 12°, cloth, 206 pp. New York: Oxford University Press, 1946. $3.00.

This small manual is designed for the general practitioner for quick reference to the therapy of the ordinary diseases. The subject matter is arranged alphabetically, and a number of symptoms, such as pain and insomnia, have been included with the diseases. The book concludes with a fifty-eight-page chapter on diet by Miss Rose Simmonds, which includes many special diet sheets and tables.

Familial Nonreaginic Food-Allergy. By Arthur F. Coca, M.D., medical director, Lederle Laboratories. Second edition, 8°, cloth, 191 pp., with 28 tables. Springfield, Illinois: Charles C Thomas, 1945. $3.75.

The author presents in this monograph a method for the diagnosis and treatment of the relatively large groups of allergic diseases, such as migraine, urticaria, gastrointestinal disorders, neuralgias and other conditions hitherto unrecognized as allergic, in which the usual cutaneous tests almost invariably fail. Under the term "idioblastic allergy" are discussed the various aspects of food allergy. The concluding chapters are devoted to a discussion of the inheritance of food allergy, its relation to hypertension, its role in the causation of the common cold and its incidence in cases of cancer of the breast.

The Early Diagnosis of the Acute Abdomen. By Zachary Cope, M.D., M.S. (Lond.), F.R.C.S. (Eng.), surgeon to St. Mary's Hospital, Paddington, and senior surgeon to Bolingbroke Hospital, Wandsworth Common. Ninth edition. 8°, cloth, 262 pp., with 38 illustrations. New York: Oxford University Press, 1946. $3.75.

The number of editions of this small manual, first published in 1921, speaks well for its popularity and soundness. This new edition contains only a few minor alterations and additions, but it should find a place in the libraries of all physicians and surgeons.

Psychological Medicine: A short introduction to psychiatry. With an appendix, "Psychiatry Associated with War Conditions." By Desmond Curran, M.B., F.R.C.P., D.P.M., psychiatrist and lecturer in psychologic medicine, St. George's Hospital; and Eric Guttmann, M.D., M.R.C.P., neuropsychiatric specialist, Emergency Medical Service. With a foreword by J. J. Conybeare, M.C., D.M. (Oxon.), F.R.C.P., physician to Guy's Hospital, London. Second edition. 8°, cloth, 246 pp., with 20 illustrations. Baltimore: Williams and Wilkins Company, 1945. $3.50.

The authors have taken advantage of the need for a new edition of their manual to make an extensive revision of their text. A number of sections and chapters have been rewritten and enlarged. The principal changes are in the sections on constitutional factors, psychopathic personalities and the affective and hysterical syndromes. A chapter has been devoted to the obsessional states, and a brief discussion of psychomatic medicine has been included in the text. A short reading list concludes the volume.

Doctors East, Doctors West: An American physician's life in China. By Edward H. Hume, M.D. 8°, cloth, 278 pp. New York: W. W. Norton and Company, Incorporated, 1946. $3.00.

Dr. Hume has written an interesting popular narrative of his twenty-five years' work in China. A graduate of Johns Hopkins University, stationed in India as an officer of the United States Public Health Service, he was persuaded by the Yale University Mission to go to Changsha, Hunan Province, in 1905, to establish a modern hospital and medical school. His work ended with the turning over of the hospital to the Chinese government in 1930. The story is valuable for the light it throws on medical practice and popular medical beliefs in China of the old days.

Journal of Gerontology: To add life to years, not just years to life. Volume I, 1946. 4°. Springfield, Illinois: Charles C Thomas, 1946. Quarterly: two parts. $6.00 a year for both parts; $3.00 a year for the nontechnical supplement.

This new journal on old age, its physiology, hygiene and diseases, the organ of the Gerontological Society, is a welcome addition to the field of special periodicals. The number of outstanding authorities comprising its editorial staff speaks well for its high standard of quality. The numbers are issued quarterly in two parts, technical and nontechnical. Each article in the former has summaries in English, French, Spanish and Russian. The nontechnical supplement is published for the general public and is written for the average reader who desires information on the subject. It contains abstracts of all the articles published in the technical part, rewritten in plain nontechnical language. It also contains discussions of current economic or social problems of the aged.

Advancing Fronts in Chemistry: A series of lectures sponsored by Wayne University under the direction of Neil E. Gordon, chairman, Department of Chemistry. Volume II: *Chemotherapy.* Edited by Wendell H. Powers, assistant professor of chemistry. 8°, cloth, 156 pp., with illustrations. New York: Reinhold Publishing Corporation, 1946. $3.25.

This series of lectures discusses the following subjects: chemotherapy in experimental tuberculosis, synthetic antispasmodics, chemistry of the sulfonamides, the antimalarial problem, organometabolic compounds and the chemotherapy of parasitic diseases. Each lecture is well documented, with pertinent references to the literature. This volume is recommended for all medical-reference collections.

The Surgical Technic of Abdominal Operations. By Julius L. Spivack, M.D., LL.D., associate professor of surgery, University of Illinois College of Medicine, senior attending surgeon, Columbia Memorial Hospital, Chicago, and attending surgeon, Oak Forest Infirmary. Fourth edition, revised. 8°, cloth, 710 pp., with 682 illustrations. Springfield, Illinois: Charles C Thomas, 1946. $10.00.

The first edition of this standard work was published in 1936, and the necessity for this fourth edition attests its popularity and soundness. The text has been revised by the elimination of some material and by the addition of new material. The type has been entirely reset. Selected bibliographies are appended to each chapter, and short historical sketches precede the discussion of each operation. The illustrations are clear and well executed. The book should prove valuable to surgeons and should be in all hospital and medical libraries.

NOTICES

NEW ENGLAND HEART ASSOCIATION

A meeting of the New England Heart Association will be held at Boston University School of Medicine auditorium, 78 East Concord Street, Boston, on Monday, March 31, at 8:15 p.m.

PROGRAM

A Note on Catheterization of the Coronary Sinus in Man and the Oxygen Content of Blood Obtained Therefrom. Drs. James W. Culbertson, Meyer H. Halperin and Robert W. Wilkins.

An Accurate Venographic Technic for the Study of Thromboembolic Disease of the Lower Extremities. Dr. George Mixter, Jr.

The Effects of Local Compression on Blood Flow in the Extremities of Man. Drs. Meyer H. Halperin, Carl K. Friedland and Robert W. Wilkins.

Adrenal Tumors and Hypertension. Drs. John B. Graham and Reginald H. Smithwick.

The Ventricular Gradient in Hypertension. Drs. Norman H. Boyer and William L. Hewitt.

Early Experiences with Total Thoracic Sympathectomy in Hypertensive Patients with Coronary Heart Disease and Angina Pectoris. Drs. Dera Kinsey and Reginald H. Smithwick.

The Effects of Sympathectomy on Certain Vasopressor Responses in Hypertensive Patients. Drs. Robert W. Wilkins and James W. Culbertson.

BOSTON CITY HOSPITAL HOUSE OFFICERS' ASSOCIATION

The April 8 lecture of the Tuesday evening lecture series of the House Officers' Association of the Boston City Hospital will be held in the new Cheever Amphitheater, Dowling Building, at 7:00. Dr. Wilder Penfield, director of the Montreal Neurological Institute, will speak on the subject, "Classification and Differential Diagnosis of the Epilepsies," which will be followed by a discussion by Dr. William G. Lennox.

GREATER BOSTON MEDICAL SOCIETY

A meeting of the Greater Boston Medical Society will be held in the auditorium of the Beth Israel Hospital on Tuesday, April 1, at 8:15 p.m. Dr. Philip E. Meltzer will speak on the subject: "Deafness: Its management and recent advances in therapy."

AMERICAN COLLEGE OF SURGEONS

The sectional meeting of the American College of Surgeons, embracing the entire northeastern region of the United States, will open on Friday morning, March 28, at 8:30, and continue through Saturday afternoon, March 29, at the Providence-Biltmore Hotel, Providence. Surgeons, members of the medical profession at large, medical students and hospital representatives from this area are invited to attend the sessions, which will begin each morning with the showing of medical motion pictures, followed by separate sessions for the medical and hospital delegates.

The scientific session for the first morning will include the following talks: "Surgery of Malignant Growths in the Neck," Dr. William F. MacFee of New York City; "Advances in Anesthesia," Dr. Ralph M. Tovell, of Hartford; "Surgery of Carcinoma of the Bladder," Dr. Samuel N. Vose, of Boston; and "Vascular Surgery," Dr. Harris B. Shumacker, Jr., of New Haven. Luncheons for physicians, surgeons and hospital representatives, followed by a discussion of the subjects covered at the morning sessions, will be held on both days.

Two panel discussions will feature the scientific sessions on the first afternoon. The first, on fractures, will be conducted by Dr. Robert M. Yergason, of Hartford, with Dr. Mather Cleveland, of New York City, and other prominent authorities serving as collaborators. The other panel will be on intestinal obstruction and will be conducted by Dr. Arthur W. Allen, of Boston.

A dinner will be held on the evening of the first day, followed by a premiere showing of a medical motion picture now under production and concluding with a reception.

The talks at the scientific session on the second morning will be as follows: "Use of Antibiotics in Surgical Practice," Dr. Frank L. Meleney, of New York City; "Surgery of Malignant Growths of the Neck," Dr. Francis D. Moore, of Boston; "Treatment of Portal Hypertension," Dr. Arthur H. Blakemore, of New York City; and "Management of Cancer of the Breast," Dr. Frank E. Adair, of New York City.

The scientific-session program for the second afternoon will consist of panel discussions on postoperative care and on carcinoma of the colon. The moderator of the first panel will be Dr. John H. Mulholland, of New York City, with Dr. James B. Blodgett, of Boston, Dr. Bliss B. Clark, of New Britain, and Dr. E. A. Rovenstine, of New York City, as collaborators. The moderator of the second panel will be Dr. Samuel C. Harvey, of New Haven, and the collaborators, Drs. Arthur W. Allen and Richard B. Cattell, of Boston.

INDUSTRIAL HEALTH MEETINGS

A conclave of combined professional personnel in industrial health work will take place at the Hotel Statler, Buffalo, New York, April 26 through May 4. These meetings will represent the thirty-second annual gathering of the American Association of Industrial Physicians and Surgeons, the ninth annual conference of the American Conference of Governmental Industrial Hygienists, the eighth annual meeting of the American Industrial Hygiene Association, the fifth annual conference of the American Association of Industrial Nurses and the fourth annual meeting of the American Association of Industrial Dentists.

The sessions will be replete with many new subjects of interest, including the following: round-table discussions for physicians and nurses; a symposium on new problems in the developments of industrial hygiene; a discussion of state codes and industrial-hygiene administration; conferences on environmental control; clinics on fractures and traumatic surgery, with a symposium on back problems; hazards incident to the use of the atomic bomb; tracer chemistry in toxicologic research; progress in the teaching of industrial medicine in American medical schools; a panel discussion on new preventive measures in industry; and a panel discussion on in-service education of the nurse in industry. Prominent speakers on important subjects will be featured at dinner sessions and at other events, such as the Cummings Memorial Lecture and the presentation of the Knudsen Award for the most outstanding contribution to industrial medicine during the past year.

Also available at this meeting will be the opportunity to inspect and study a splendid group of scientific and technical exhibits, with the most recent developments and medical-department accessories.

Further details and a copy of the preliminary program may be secured by writing to Dr. Edward C. Holmblad, American Association of Industrial Physicians and Surgeons, 28 East Jackson Blvd., Chicago 4. All hotel reservations are made by the Housing Bureau, Buffalo Convention and Tourist Bureau, Incorporated, 602 Genesee Building, Buffalo, New York.

AMERICAN PUBLIC HEALTH ASSOCIATION

The seventy-fifth annual meeting of the American Public Health Association will be held in Atlantic City, October 6–10. The scientific program and the exhibits will emphasize progress in public health during the last seventy-five years. The following related organizations will participate in the meeting: American School Health Association; Association of Maternal and Child Health Directors; Association of Reserve Officers of the United States Public Health Service; Association of State and Territorial Health Officers; Conference of Municipal Public Health Engineers; Conference of Professors of Preventive Medicine; Conference of State and Provincial Public Health Laboratory Directors; Conference of State Directors of Health Education; Conference of State Directors of Public Health Nursing; Conference of State Sanitary Engineers; National Committee of Health Council Executives; and Public Health Cancer Association.

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Thursday, March 27

Friday, March 28

*9:00–10:00 a.m. The Effect of Carotid Sinus Stimulation on Anginal Pain, and Other Considerations. Dr. Samuel Levine. Joseph H. Pratt Diagnostic Hospital.

*10:00 a.m.–12:00 m. Medical Staff Rounds. Peter Bent Brigham Hospital.

Monday, March 31

*12:15–1:15 p.m. Clinicopathological Conference. Peter Bent Brigham Hospital.

8:15 p.m. New England Heart Association. Auditorium, Boston University School of Medicine.

Tuesday, April 1

*12:15–1:15 p.m. Clinicoroentgenological Conference. Peter Bent Brigham Hospital.

8:15 p.m. Greater Boston Medical Society. Auditorium, Beth Israel Hospital.

Wednesday, April 2

*11:00 a.m.–12:00 m. Medical Clinic. Amphitheater, Children's Hospital.

*12:00 m. Clinicopathological Conference. (Children's Hospital.) Amphitheater, Peter Bent Brigham Hospital.

*2:00–3:00 p.m. Combined Clinic by the Medical, Surgical and Orthopedic Services. Amphitheater, Children's Hospital.

*Open to the medical profession.

March 25. Boston City Hospital House Officers' Association. Page 382, issue of March 6.

March 26. Tufts Medical Alumni Association. Page 382, issue of March 6.

March 27. Winthrop Community Hospital. Page 340, issue of February 27.

March 28 and 29. American College of Surgeons. Notice above.

March 31. New England Heart Association. Page 455.

April 1. Greater Boston Medical Society. Notice above.

April 8. Harvard Medical Society. Amphitheater, Peter Bent Hospital. 8:00 p.m.

April 8. Boston City Hospital House Officers' Association. Notice above.

April 10. Physiotherapy, Its Practical Application and Its Limitations. Dr. Franklin P. Lowry. Pentucket Association of Physicians. 8:30 p.m. Haverhill.

April 15–18. Postgraduate Institute. Page 382, issue of March 6.

April 26–May 4. Industrial Health Meetings. Notice above.

April 28–May 2. American College of Physicians. Page 206, issue of August 8.

(*Notices continued on page xxi*)